A STUDY ON

VALI AZHAL KEEL VAYU

Dissertation Submitted To

THE TAMIL NADU DR.M.G.R Medical University

Chennai – 32

For the Partial fulfillment for The Award of Degree of

DOCTOR OF MEDICINE (SIDDHA)

(Branch – III, SIRAPPU MARUTHUVAM)

DEPARTMENT OF SIRAPPU MARUTHUVAM

Government Siddha Medical College

Palayamkottai – 627 002

September - 2008

INTRODUCTION

Siddha system of Medicine is a traditional one with prestigious background of Tamil culture. It's perhaps the earliest medical science that laid stress on positive health, a harmonious blending of physical, mental, social, moral and spiritual welfare of an individual.

Medicine is not merely a science but an art as well. The practice of siddha medicine deals not merely with the eternal body of man but also with the inner man or soul. The art of medicine is based on Truth and as much as such it is divine art not to be prostituted for base purposes of money.

The employment of strong will, benevolence, charity, patience etc is the principle corner stone in the practice of medicine.

The siddhars were further the greatest alchemist in ancient times. They were men of highly cultured intellectual and spiritual faculities to be more valuable than any.

"கருந்துறவே வாதி மகன் வைத்தியம் செய்யில்
கசடறவே நோயாளி குருவா யென்னி
கருந்துறவே மருந்துக்கு பக்தி வேணும்
கைகண்ட வைத்தியரை அறிந்து நன்றாய்
கருந்துறவே மனதுவர நடக்க வேணும்
கழுதையைப்போ லிருந்தவர்க்கு பலியா தொன்றும்
கருந்துறவே யிவையறிய சித்தர் தானும்
காட்டினார் அவர் மனம் போல் நடந்து கொள்ளே"

– நாடி சாஸ்திரம்

The character of physician may act more powerfully upon the patient than all drugs employed and likeness it is verified among patients that a strong belief, undoubted faith and deep love for physician conduce much towards their health even more than the medicine itself.

Thirumoolar has quoted

“ மறுப்பது உடல் நோய் மருந்தெனலாகும்
மறுப்பது உள நோய் மருந்தெனலாகும்
மறுப்பது தினி நோய் வாராதிருக்க
மறுப்பது சாவை மருந்தெனலாமே”

– திருமூலர் எண்ணாயிரம்

Siddha medicine in the one which prevents the body and mind from a diseased condition by curing the present disease and by working also as a rejuvenating process for further prevention of the same by treating whole body as a whole.

According to manuscripts as well as the evidences found out disease may occur due to the derangement of either the external or internal factors.

The internal factors include illness occurring due to the disturbance of three humours, the seven thathus, the three Gunas and the malas the secretive and excretive

The external factors include seasonal changes or climatic variations or unlikable things like drug abusing and external stress ending in psycho somatic disorders.

The author has taken the disease Vali azhal keel vayu as per dissertation topic on the basis of siddha concept, on the course of the disease, treatment, prognosis and dietetic aspects.

AIM AND OBJECTIVES

Vali azhal keel vayu is a universal disease affecting 3% of the total population, involving poor as well as rich people and causing disabilities and deformities. Vast studies on this disease and as per the opinions from the concerned specialists reveal that still there is not a total curative therapy for this disease. The signs and symptoms of the disease are correlative with Rheumatoid arthritis. The author has selected this disease and treated with the help of **பறங்கிப்பட்டை இரசாயனம் (Parangipattai Rasayanam)** 1-2 gms 2 times Morning and Evening after food a day internally and **கீல்வாதத்திற்கு ஐந்தென்ணெய் (Keelvathathuku Iynthennai) externally.**

- To collect authentic measures and review the ideas mentioned in ancient siddha literature about the disease.
- To study the clinical features of the disease vali azhal keel vayu.
- To review the altered Tridosha or mukkutram and changes in the physiology as per siddha aspect.
- To study the disease Vali azhal keel vayu on the basis of Udal thathu, paruva kaalam, food, taste, age, sex,socio-economic status, ennvagai thervu, neerkuri and neikuri.
- To expose the unique diagnostic procedure mentioned in siddha literature for the disease Vali azhal keel vayu.
- To diagnose the disease on the basis of modern parameters
- To have a detailed analysis to prove the clinical efficacy of the drugs through the pharmacological and biolchemical analysis.

The work is to be carried out by having a clinical trial by using **பறங்கிப்பட்டை இரசாயனம் (Parangipattai Rasayanam)** as internal drug and **கீல்வாதத்திற்கு ஐந்தென்ணெய (Keelvathathuku Iynthennai)** as external drug on 30 inpatients with the disease Valiazhal keelvayu.

SIDDHA LITERATURE

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5. $\leftrightarrow \varnothing \rfloor \oplus \cap' \div \leq \rfloor \oplus \Upsilon^{\text{TM}}$
6. $\oplus \cap' \geq \Re \div \leq \rfloor \oplus \Upsilon^{\text{TM}}$
7. $\leftrightarrow \varnothing \rfloor \geq \Re \div \leq \rfloor \oplus \Upsilon^{\text{TM}}$
8. $\geq \Re \oplus \cap' \div \leq \rfloor \oplus \Upsilon^{\text{TM}}$
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$\clubsuit \hookleftarrow \Upsilon \backslash \oplus \prod \rfloor \ \oplus \varnothing'$ **(Aetiology)**

$\mathbf{2}\_ \mid \cdot, \aleph \varnothing \lceil \div \oplus \varnothing \lceil \div \{ \div \rfloor, \subseteq \rceil \vee \mid ® \cup \Uparrow \lfloor \hookleftarrow' \blacklozenge \otimes \leftarrow \div ' \Re \ \copyright \} \vee \rfloor$
$\copyright \lceil \subset \rceil \cup \int — ' \rfloor \ \Im \Upsilon \vee \aleph \Upsilon \in \ \mathbf{2}\_ \rangle \ldots \Upsilon \lceil \div ' \ \clubsuit \hookleftarrow \Upsilon \blacklozenge \Re \ '' \rceil \aleph \in \int \angle \div ' \cup \angle .$

$\subseteq \rceil \vee \mid ® \cup \Uparrow \lfloor \hookleftarrow' \blacklozenge \otimes$

$\forall \oplus \Upsilon — \oplus \vdash — \supset \div \Upsilon \otimes \clubsuit \Im \clubsuit — \Upsilon \ f \oplus \} \supset ' \rfloor$

$$\mathfrak{I}\prod\cdot\div'\}\cup\leftarrow\supset'\div]\div\ldots\ \mathfrak{I}\Upsilon\text{—}\mathcal{J}$$

$$\longleftarrow\blacklozenge\supset\mid\ \aleph\equiv'\clubsuit\Re\Upsilon\in\div\Upsilon\ |\int\text{—}'\blacklozenge\div\text{—}\}\supset'\rangle$$

$$\leftrightarrow\ldots\prod\clubsuit\mathfrak{I}\ \mathfrak{I}]\cup\ \mathfrak{I}\Upsilon\text{—}\{\div\rfloor\text{—}\}\supset'\rangle$$

$$\clubsuit\aleph\Upsilon\div\clubsuit\oplus\equiv\mathfrak{I}'\lceil\div'\}\cup\div\Upsilon\otimes\mathfrak{I}\Upsilon\subset\mathcal{J}\ \forall$$

$$-\ ™\div'\equiv'\int\text{—}\Upsilon\mathfrak{I}\mid'$$

$$\oplus\cap'\ \clubsuit\lrcorner\Upsilon\Re\Upsilon\supset\angle\leftarrow\supset'\ ©\text{—}\rangle\div\Upsilon\ |\int\text{—}'\blacklozenge\div\ \mathfrak{I}\Upsilon\text{—}\{\div\cap'\rangle\leftrightarrow\text{—}'\div\mathfrak{I}\Upsilon\div\ \div\Upsilon\mid\mid$$

$$\aleph\in\mathcal{J}\,.$$

$$\forall\aleph\angle\mathfrak{I}\int\blacklozenge\text{—}\ ®\lceil\div\blacklozenge\oplus\lceil\subset\mathcal{J}\ \ \aleph\Upsilon\Leftrightarrow\mathfrak{I}'\div\lceil\ \div\Upsilon™\mathcal{J}$$

$$©\angle\clubsuit\oplus\supset'\otimes'\rceil\ ®\ \oplus'\rceil\lrcorner\leq]\mid©]\vee\mathcal{J}\ -\div\angle f\mathfrak{I}\supset$$

$$\oplus]\vee\mathcal{J}\ \div\aleph\mathcal{J}\bullet\subset\mathcal{J}\ \oplus\Upsilon™\mathfrak{I}'\subset\mathcal{J}\ \oplus\Upsilon\ |\mathfrak{I}\Upsilon\ \int\int||$$

$$\subset]\cup\lrcorner\otimes'\lceil\clubsuit\div\text{—}'f\text{—}\}\ \clubsuit\cup\Upsilon\angle.\forall$$

$$-\equiv'\int\text{—}\ \mathfrak{I}\prod\int\angle\oplus\Upsilon\{\div\lfloor\subseteq\prod\lceil\div\mathcal{J}\,.$$

$$©\angle\clubsuit\oplus\supset']\ \div\Upsilon\otimes\int\text{—}'\rangle,\Uparrow\wp'\Re\ f\oplus\mid\aleph\int\text{—}'\}\div\Upsilon\wp\mid\mathfrak{I}\Upsilon\div f\aleph\prod\mathcal{J}\ \oplus\Upsilon$$

$$\wp'\Re\Upsilon\ \lrcorner\leq\mid\leftarrow\oplus'\Re\Upsilon\lceil\div\mid\aleph\square\in\ 2\_\otimes\div\int\text{—}'\rangle\oplus\cup\square\equiv'\ \lrcorner'\otimes\cdot\mathcal{J}\,.\leftrightarrow\angle\ \clubsuit\aleph\Upsilon\rangle\ \lrcorner\mathfrak{I}$$

$$\angle\ 2\_\ldots\otimes'\rangle\oplus\cup\square\equiv''\rceil\aleph\square\in\oplus\cap'\ \clubsuit\lrcorner\Upsilon]\oplus\prod\oplus\text{—}]\subset''\angle\oplus\Upsilon\div'\ni\cup\angle.$$

$$\text{ƍ}\_\,|\,.$$

$$\forall\oplus\cap'\,\text{—}\prod\div\Upsilon\rangle\div'\varnothing\langle\subset\oplus\blacklozenge\,\wp\oplus'\otimes\Upsilon\,\text{—}\Re'\otimes\rfloor\,\clubsuit\div\Upsilon\blacklozenge\varnothing$$

$$\copyright\cap'\,\text{—}\Re'\,|\,\clubsuit\aleph\Upsilon\}\Im'\subset\lceil\subset\copyright\blacklozenge\cup\Re'\otimes\Upsilon\cdot\rangle\supseteq\,\clubsuit\div\Upsilon\ldots\rfloor$$

$$\subset\cap'\vdash\!\!\text{—}\prod\oplus\cap'\Re'\rceil\,\clubsuit\cup\div\langle\,\subset\supset'\,|\,\circledR\cup\cdot\otimes\oplus\rfloor\,f\aleph\rangle\supseteq\,|$$

$$\subset\cap'\text{—}\prod\copyright\Re\lceil\div\rfloor\,\,f\aleph\rceil\,\clubsuit\cup\Upsilon\,|\div\supseteq f\equiv\Re\rfloor\div\prod\oplus'\Re\Upsilon\Im\Upsilon\rfloor.\forall$$

$$-\equiv\aleph\Upsilon\aleph\text{—}'\,\blacklozenge\div\clubsuit\Re\in$$

$$\oplus\Upsilon\text{—}\subset\rceil\cup\int\blacklozenge\text{—}\,\Im'\blacklozenge\div\,|\,\aleph\in\int\text{—}\lceil\,\Leftarrow\supseteq\Re\div'\varnothing\langle\subset\oplus\blacklozenge\div\div\rfloor,\subset\cap'\,|\lfloor\equiv'\blacklozenge$$

$$\Re$$

$$\text{—}\wp\lceil\Leftarrow\supseteq\Re\,\,\aleph\text{—}\Upsilon\,\|\int\text{—}\langle\div\rfloor\Im'\subset\text{—}'\Re\Upsilon\div\text{ƍ}\_\rangle\,|\,\rfloor\subset\cap'\,|\,\lceil\text{—}\div\Upsilon\rceil\cup'\rfloor$$

$$\rfloor\prod\int\text{—}\rfloor,\,\Im\blacklozenge\varnothing\Re'\rfloor\,\,\hookleftarrow\blacklozenge\supset\text{—}\rfloor,\,\,\aleph\supset'\lceil\div\Upsilon\rceil\vee\,\clubsuit\Im\clubsuit\otimes\oplus'\neg\text{—}\rfloor,\Im\blacklozenge\otimes\,|$$

$$\aleph'\wp\clubsuit\text{—}\equiv\langle\div\cap'\rfloor\oplus\equiv'\int\text{—}\rfloor,\,\Im\rceil\vee\rfloor\,\,\aleph\,\wp\rfloor\,\aleph\blacklozenge\wp\Re'\}\div\Upsilon\,\wp\,|\,\Im\Upsilon\div\cdot\rfloor\,\,\clubsuit\text{—}\Upsilon\}\vee\rfloor\,.$$

$$\square f\text{—}\Upsilon\varnothing'\rfloor\,f\aleph\vee\blacklozenge\div\,|\,\circledR\lceil\,\div\Upsilon\,\|\int\text{—}\rfloor\,\angle\oplus\,\|\int\text{—}\rfloor\,\oplus'\,|\subseteq\div'\Leftrightarrow|\,\clubsuit\equiv\Upsilon\vee\rfloor$$

$$\aleph\blacklozenge\varnothing\Re\text{—}\Upsilon\rfloor\,\,\oplus\wp\subset\Im\rceil\,\blacklozenge\cup\,|\,\,\blacklozenge\aleph\,\lceil\text{—}'\blacklozenge\supset\Re\prod\lceil\text{—}'\supset\Upsilon\surd\rfloor$$

$$\pm\varnothing'\rfloor\,f\aleph\cup\,|\,\,\aleph\div\surd\cup\langle\div'\,\rfloor\,\wp\oplus'\supset'\surd\cup\langle\div\Upsilon\text{———}\Upsilon\surd\rfloor$$

$$\Im\blacklozenge\varnothing\hookleftarrow'\div\,|\subset\varnothing\otimes'\supset\Upsilon\clubsuit\otimes\,\oplus\Upsilon\text{—}\langle\,\clubsuit\div\Upsilon\aleph'\lceil\subset\lceil\text{—}\Upsilon\clubsuit\supset\square$$

$$-\,\text{™}\div'\equiv'\,\lceil\text{—}\Upsilon\Im\,|\,'\,(231)$$

$$\div\equiv|\,\circledR,\angle\oplus\,|\,|\,\circledR,\div\Upsilon\,|\,|\,\circledR,\aleph\text{—}\Upsilon\,\|\int\text{—}\langle\div\blacklozenge\cap\,\Im'\subset\text{—}'\Re\Upsilon\div\text{ƍ}\_\rangle\,|\,\rfloor,\,\aleph\blacklozenge$$

$$\varnothing\Re\clubsuit\equiv\Upsilon\vee,\oplus\wp\subset,\text{—}'\blacklozenge\supset\text{ƍ}\_\rangle\,|\,\rfloor,\,\aleph\div\otimes'\rfloor\,\Diamond\langle\div'\,\rfloor\,\wp\cdot\,\oplus'\varnothing'\int\text{—}\rfloor\,\leftarrow\div'\Re$$

$$\div\Upsilon\wp\,|\langle\div\cap\Upsilon\surd\rfloor\,\,\oplus\Upsilon\text{—}\rfloor\,\Im'\subset\aleph\in\rfloor\,.$$

$$\aleph\varnothing\lceil\div\oplus\varnothing\lceil\div\langle\div\rfloor$$

$$\forall f\oplus\rangle\Re'\otimes'\rfloor\,\hookleftarrow\ldots\lceil\blacklozenge\div\Re\Upsilon\surd\rfloor\,\,\Im'\div\int\text{—}\rangle\,|\leq|\subset\supseteq\lceil\blacklozenge\div\Re\Upsilon\surd\rfloor$$

$$f\equiv\rangle\Re'\blacklozenge\varnothing\,\Im\div\cap'\blacklozenge\,\wp\lfloor\,\,\clubsuit\equiv|\,\lceil\text{—}\Leftrightarrow\aleph\,\oplus'\lceil\blacklozenge\div\Re\Upsilon\surd\rfloor$$

$$\blacklozenge\aleph\Re\clubsuit\supset\,\rangle\blacklozenge\Im\Re\Upsilon\surd\rfloor\,\,\aleph\Upsilon\div\rceil\div\Upsilon\rangle\text{—}'\}\blacklozenge\div\Re\Upsilon\surd\rfloor$$

$$\blacklozenge\text{—}\Re\clubsuit\otimes\oplus\Upsilon\text{—}\clubsuit\wp\Upsilon\div\jmath\equiv\supset'\lceil\subset\jmath f\Im\}\cup\cup'\lceil\angle f\div\Upsilon\rfloor\clubsuit\cap\forall$$

$$-\clubsuit\text{—}\blacklozenge\wp\Re\,|\oplus\Upsilon\div\ldots\jmath\,.$$

$$\Uparrow\wp'\Re\,f\oplus\,|\,\aleph\int\text{—}'\jmath\leftrightarrow\text{—}'\div f\text{—}\Upsilon\blacklozenge\otimes\cdot\,\lrcorner\ldots\lceil\blacklozenge\div\Re\Upsilon\sqrt{\jmath}\,,\leftrightarrow\text{—}'\div\text{—}\rangle\,|$$

$$\leq|\subset\supseteq\lceil\blacklozenge\div\Re\Upsilon\sqrt{\jmath}\,,\leftrightarrow\text{—}'\div\leftrightarrow\cap\cdot\,\aleph\Upsilon\div\rceil\div\Upsilon\rangle\equiv\Upsilon\,|\,\aleph'\in\oplus\text{—}\Upsilon\sqrt{\jmath}\,,\oplus\cap'\,\clubsuit\lrcorner\Upsilon\rangle$$

$$''\rceil\aleph\in\jmath\,.$$

$$\square\div\Upsilon\,|\,\clubsuit\oplus\Im'\div\cdot\rangle\ldots\Upsilon\sqrt{\}\div\prod\angle\aleph\square\supseteq\supset'\oplus'\square\ldots\sqrt{\jmath}$$

$$\Im\Upsilon\supset\blacklozenge\supset\Re\Upsilon\,|\div\rangle\,\clubsuit\Im\Upsilon\div\Im\cup\lceil\div'\Leftrightarrow\Im'\subset\int\text{—}'\square\ldots\sqrt{\jmath}$$

$$\leftarrow|\oplus\Im\otimes\{\div\ldots\jmath\,\blacklozenge\Im\,\Re\{\neq\clubsuit\supset\oplus'\ldots\Upsilon\text{——}\Upsilon\sqrt{\jmath}$$

$$\oplus\Upsilon\Leftrightarrow\text{—}\}\,\Im\ldots\lrcorner\jmath\otimes\Upsilon\clubsuit\cap\oplus\Upsilon\text{—}\{\clubsuit\div\Upsilon\aleph'\lceil\subset\{\div\Upsilon\clubsuit\,|\,\square$$

(232)

$$\leftrightarrow\text{—}'\div\leftrightarrow\cap\cdot\,\text{ᴥ}\rangle\,|\jmath,\aleph\square\supseteq\supset'\div'\ldots\int\text{—}\jmath,\leftarrow|\oplus\jmath\,\leftrightarrow\text{—}'\div\wp'\int\text{—}\jmath\leftarrow\div'\Re\oplus\rceil\cup\Upsilon\sqrt{\jmath}$$

$$\square\aleph\Upsilon\wp'\supset'\rceil\,\aleph\Re\,|\,\aleph\square\ldots\sqrt{\jmath}\,\aleph\otimes\prod\ldots\}\,\clubsuit\div\Upsilon\aleph'\int\text{—}\Upsilon\sqrt{\jmath}$$

$$\div\Upsilon f\wp\supset\lceil\div\prod\div'\clubsuit\Re\Upsilon\supseteq\lceil\div\neg\Im\wp\int\angle\wp\int\text{—}'\supset\Upsilon\sqrt{\jmath}\div$$

$$''\,|f\aleph\vee\text{—}\supset\angle f\lrcorner|\equiv'\}\,\Im'\div\int\angle\lceil\div\Im\blacklozenge\ldots\int\text{—}'\square\ldots\Upsilon\sqrt{\jmath}$$

$$\aleph\Upsilon\wp'\Re\div\Upsilon\rceil\cup'\supset\Upsilon\sqrt{\jmath}\,\aleph\ldots\wp'\Leftrightarrow\jmath\,\oplus\Upsilon\text{—}\{\div\Upsilon\notin\jmath\,\square$$

(233)

$$\aleph\Re\jmath\,,\pm\jmath\otimes\wp'\ldots\copyright\jmath\,\clubsuit\div\Upsilon\aleph\jmath\,f\div\Upsilon\rfloor\cap\jmath,\Im'\subset\text{—}'\Re\Upsilon\div\times\ldots\jmath,\Im'\subset\int\text{—}\angle\lceil\div\jmath\text{—}'\supset\copyright\jmath\,\text{ᴥ}\ldots\otimes'\}\,\clubsuit\Im\jmath\div\Upsilon\rceil\vee|\aleph\ldots\jmath\,\clubsuit\aleph\Upsilon\}\cup\oplus\rceil\cup\Upsilon\sqrt{\jmath}$$

$$\square\div\Upsilon\otimes\{\div\rangle\,\Im\Upsilon\cup'^{\text{™}}\rangle\notin\{\div\Upsilon\wp'\Re\int\text{—}\Upsilon\sqrt{\int\text{—}\rangle}\,|\leq|$$

$$\equiv\Upsilon\otimes\clubsuit\oplus\Re\prod\int\text{—}'\supset\Upsilon\sqrt{\int}\equiv\int\text{—}'\Re'\sqrt{\square}\div\Upsilon\,|\int\text{—}\Upsilon\sqrt{\jmath}$$

$$\clubsuit\div\Upsilon\otimes\Im\Upsilon\jmath\,\circledR\cap'\,|\circledR\,f\lrcorner\rangle\blacklozenge\Re\lceil\subset\blacklozenge\cup\oplus\cup\oplus\prod\int\text{—}'\supset\Upsilon\sqrt{\jmath}$$

$$\oplus\Upsilon\otimes\oplus\Upsilon \mid \copyright\blacklozenge\otimes\lrcorner\jmath\otimes\Upsilon\clubsuit\cap \oplus\Upsilon\text{—}\copyright\rceil\aleph\oplus'\lceil\subset\} \div\Upsilon\clubsuit \mid \square$$

(234)

$$\subset\cap' \mid \div\Upsilon\rceil\cup'\jmath \text{ ᵹ}\square\div\Upsilon \mid \int\text{—}'\prod\int\text{—}\jmath, \circledR\cap' \mid \circledR, f\lrcorner\rangle, \text{ ᵹ} \mid \oplus'\jmath\ \Im'\subset\text{—}'\Re\Upsilon \div \clubsuit\equiv \lvert\int\angle\lceil f\div\Upsilon\rfloor\cap\jmath, \div\Upsilon\otimes\jmath\ \text{—}\oplus\cup' \text{ ᵹ}\rangle \mid \jmath \leftarrow\div'\Re\ \div\Upsilon\wp \mid\{\div\cap\Upsilon\surd\jmath\ \oplus\Upsilon\text{—}\ \clubsuit \div\Upsilon\aleph\jmath\ \text{ ᵹ}\rangle\ldots\Upsilon\oplus\text{—}\Upsilon\div\ \aleph\wp\wp\Upsilon\equiv\clubsuit\equiv\div\wp\int\text{—}'\jmath\ f\equiv\Upsilon\jmath\otimes \mid \aleph\square\in\rfloor\cap\angle.$$

$\oplus\Upsilon$— ♣↵Υ⟩⌈÷$\Upsilon\supset$⌋$\Re\jmath$® (Χηαραχτεριστιχ φεατυρεσ οφ ςατηαμ)

$$\forall\oplus\Upsilon\text{—}\clubsuit\Im\ \div\text{—}'\int\text{—}\ \clubsuit\aleph\Upsilon\angle\ \oplus\Upsilon^{\text{™}}\cdot f\Im\neg\jmath\ \circledR\{\ \div\rangle\not\subset \mid$$

$$\oplus\Upsilon\text{—}\clubsuit\Im\ \div\text{—}'\int\text{—}\ \clubsuit\aleph\Upsilon\angle\ \oplus\Upsilon^{\text{™}}\Im\ \int\text{—}'\in \mid\ \equiv\}\supset'\ \clubsuit\text{—}\Upsilon\square\jmath$$

$$\oplus\Upsilon\text{—}\clubsuit\Im\ \div\text{—}'\int\text{—}\ \clubsuit\aleph\Upsilon\angle\ \oplus\jmath\surd\ldots\}\ f\Im\otimes'\ \int\angle\ f\div\Upsilon\jmath\surd\jmath\ \forall$$

$$-\leftrightarrow\div\int\text{—}'\Re \mid \equiv'\div'\lfloor\equiv\Upsilon\wp\int\supset\text{—}\leq\aleph\jmath$$

$$\oplus\Upsilon\text{—}\jmath\ \Im'\subset\jmath\ \clubsuit\aleph\Upsilon\angle\ \oplus\Upsilon^{\text{™}}\ \Im'\subset\jmath\ .\equiv\}\supset'\ \clubsuit\text{—}\Upsilon\ldots\jmath\ \clubsuit\aleph\Upsilon\}\cup\ \aleph\otimes \oplus'\Re\Upsilon\text{—}'\div\rfloor\oplus\int\angle\ \clubsuit\equiv\prod\jmath\ .\ \text{ ᵹ}\ldots\jmath\ f\Im\otimes'^{\text{™}}\jmath\ .$$

$$\forall\oplus\Upsilon\text{—}\oplus\leq\vee\leftrightarrow\}\supset\Im'\cup\{\div\Upsilon\angle\ \div\in \mid \circledR\rangle\ldots\Upsilon\jmath\ \oplus\rangle \mid \copyright\rangle\ldots\Upsilon\jmath$$

$$\clubsuit\Im\Upsilon\angle\div\square\subset f\wp\Upsilon\div\jmath\ \subseteq\wp\copyright\rangle\ldots\Upsilon\ \Im'\prod\Im\surd\Im\Upsilon\ \copyright\cup\{\div\Upsilon f\text{—}\}\vee\jmath$$

$$\times\angle\text{—}\wp'\Re\ \oplus\Upsilon\text{—}\Im\supset\otimes\Upsilon\subset\ \lrcorner\in\lceil\div\copyright\rangle\ldots\Upsilon\jmath\ f\aleph\Upsilon\prod\rfloor\div\cap\Re \mid\int\text{—}$$

$$\text{—}\leq f\text{—}\supset\clubsuit\oplus\ \lrcorner\wp\jmath\ \aleph'\int\angle\equiv\lceil\angle\div\rfloor\ \clubsuit\text{—}\Upsilon\vee\{\div\in\lceil\subset\int\text{—}'\supset\copyright\ \int\text{—}\Upsilon\clubsuit\supset$$

$$\forall$$

$$-\ \clubsuit\text{—}\blacklozenge\ \wp\Re \mid \oplus\Upsilon\div\ldots\jmath$$

$$\oplus\Upsilon\text{—}\jmath\ \Im'\subset\jmath\ \clubsuit\aleph\Upsilon\angle\ \aleph\equiv'\Re'\}\blacklozenge\Im,\ \text{ ᵹ}\ldots\jmath\div\in \mid \circledR,\subseteq\wp\jmath\ ,\rfloor\prod\Im\jmath,\ \text{ ᵹ}\cup\lceil\div\Im'\}\blacklozenge\Im,\ \text{ ᵹ}\ldots\jmath\ \lrcorner\in\lceil\div\jmath\ ,\lrcorner\wp\jmath\ \circledR\text{—}\cap\lvert\lfloor\equiv',\equiv\lceil\angle\div\rfloor\ \clubsuit\text{—}\Upsilon\vee\jmath\ \subset\blacklozenge\ldots\text{—}\jmath\ ,\clubsuit\aleph\Upsilon\}\cup\subset\cup'\subset\lvert\{\div\rfloor\ \ \clubsuit\text{—}\Upsilon\}\vee\jmath\ .$$

$$\forall\text{—}\lceil\div\oplus\Upsilon^{\text{™}}\ \clubsuit\div\Upsilon\aleph'\int\text{—}\Upsilon\jmath\equiv\int\angle\blacklozenge\cap\int\angle\Uparrow\blacklozenge\otimes\clubsuit\lrcorner\Upsilon\oplus\Upsilon$$

$$\Im' \lceil \div f \div \Upsilon \square \ldots \Upsilon \oplus' \oplus' \square \ldots \{ \ f \div \wp'^{\text{™}} \ \Im \otimes \{ f \div \square \in \jmath$$

$$\times \lceil \div \hookleftarrow \wp \jmath \circledR \text{—} \Upsilon \} \ \copyright \ldots \{ \subset \Im \otimes \mid \lceil \angle \oplus \Upsilon \rangle \hookleftarrow \leq \prod \cup' \oplus \prod \jmath$$

$$\Im' \lceil \div \subset \cap' \prod \jmath \ \hookleftarrow \in \lceil \div \Im \Upsilon \rangle \ \clubsuit \Im \supset' \subset \cap \cup' \oplus \prod \{ \div \Upsilon \clubsuit \mid \forall$$

$$- \clubsuit \text{—} \blacklozenge \wp \Re \mid \oplus \Upsilon \div \ldots \jmath$$

$$\oplus \Upsilon \text{—} \jmath \ \Im' \subset \jmath \ \clubsuit \aleph \Upsilon \angle \copyright \square \in \lceil \div \cap' \jmath \oplus \otimes', \Im' \subset \lceil \text{—} f \div \Upsilon \square \ldots \Upsilon \oplus', \Im \otimes \lfloor \equiv' \lceil \div \jmath \hookleftarrow \wp \jmath \circledR \int \text{—} \cap \mid \lfloor \equiv', \oplus \Upsilon \rangle \hookleftarrow \leq \sum \wp \jmath, \Im' \div \subset \cap' \mid \lfloor \equiv', \textbf{ᴥ} \ldots \jmath \hookleftarrow \in \{ \div \jmath \ \clubsuit \aleph \Upsilon \} \cup \subset \cup' \ \subset \mid \{ \div \rfloor \clubsuit \text{—} \Upsilon \} \vee \jmath .$$

$\oplus \cap' \leftrightarrow \varnothing \jmath \div \leq \jmath \oplus \Upsilon^{\text{™}}$

$$\clubsuit \hookleftarrow \Upsilon \rangle \subset \cup' \subset \mid \{ \div \rfloor$$

$$\forall \oplus \Upsilon \text{—} \aleph' \int \text{—} \div \leq \jmath \oplus \Upsilon^{\text{™}} \oplus' \} \oplus \prod \{ \subset \cup' \equiv \Upsilon \rceil \cup \lceil \ \clubsuit \div \cap \Upsilon \rangle$$

$$'' \text{—} \Im \Upsilon \mid \Im \int \text{—} \clubsuit \Im \mid \aleph \jmath \ \rfloor \blacklozenge \wp \lfloor \equiv \surd \jmath \ \oplus \Re' \rceil \cup' \rceil \ \div \Upsilon \notin \jmath$$

$$\times \text{—} \prod \{ \ \subset \int \text{—} \jmath \oplus \leq \lceil \div \jmath \ \times \rangle \text{—} \otimes' \jmath \pm \wp' \lfloor \equiv \surd \rangle \ldots \Upsilon \jmath$$

$$\div \Upsilon \text{—} \vee \copyright \cup \lceil \div \Im' \} \blacklozenge \Im \div \Upsilon \rangle \lfloor \equiv \surd \jmath \ \div \Upsilon \notin \{ \ \div \rangle \ldots \Upsilon \rangle \forall$$

$$- \equiv \aleph \Upsilon \aleph \text{—}' \ \blacklozenge \div \clubsuit \Re \in .$$

$$\Im \int \text{—} \jmath \, , '' \mid \aleph \jmath \, , \oplus \Re' \vee \rfloor \blacklozenge \wp \lfloor \equiv \jmath, \subset \int \text{—} \jmath, \oplus \leq \lceil \div \jmath \, , \div \Upsilon \rangle \lfloor \equiv \jmath,$$

$$\textbf{ᴥ} \cup \lceil \div \Im' \} \blacklozenge \Im \ \clubsuit \aleph \Upsilon \} \cup \subset \cup' \subset \mid \{ \div \rfloor \div \Upsilon \notin \jmath .$$

$$\forall \blacklozenge \div \div \Upsilon \rfloor \, f \aleph \Upsilon \prod \int \angle \div \cap' \rfloor \div \wp \in \div \square \supseteq \, \clubsuit \Im \supset' f \Re \rfloor \otimes \Upsilon \rfloor$$

$$\text{—} \supseteq \int \angle \, ® \rangle \mid \Upsilon \rfloor \, \oplus \Upsilon \text{—} \aleph' \int \text{—} \Uparrow \blacklozenge \otimes \pm \supset \mid \aleph \in \rfloor \, \forall$$

$$- \oplus \Re' \int \text{—}' \Re \equiv \Upsilon \, \wp \equiv \nmid \div' \, \wp \div \rfloor$$

$$\forall \oplus \cap' \Re \angle \div \notin \oplus' \blacklozenge \ldots \, \hookleftarrow' \rceil \subset \Im \Upsilon \Re' \}$$

$$\div \notin \lceil \div \rfloor \oplus \leq \nmid \div \sqrt{} \rfloor \subset \int \text{—} \sqrt{} \, \lceil \clubsuit \text{—} \Upsilon \} \vee \rfloor$$

$$\div \Upsilon \rceil \, f \aleph \prod \oplus' \, \wp \otimes' \blacklozenge \ldots \int \clubsuit \text{—} \Upsilon \} \cup' \mid \aleph \, \wp \cdot \rfloor$$

$$\oplus \cap' ™ \, \Im \supset \sqrt{\nmid} \, \div \otimes \lceil \angle \equiv' \supset \oplus \otimes' \}$$

$$\text{ಲ} \ldots \rfloor \, ® \div \supset f \otimes \supset \lceil \, \div \Upsilon \, \lceil \text{—} \rfloor \, \clubsuit \hookleftarrow \Upsilon \text{—} \rfloor$$

$$\div \Upsilon \rceil \vee \oplus \leq \equiv' \vee \rfloor \, f \aleph \Upsilon \prod \int \text{—} \otimes \prod \wedge \rfloor$$

$$\leftrightarrow \wp' \text{—}' \} \oplus \leq \nmid \div \sqrt{} \rfloor \, \clubsuit \text{—} \Upsilon \} \cup' \in f \Im \} \aleph$$

$$\text{—} \supset' \mid \aleph \in \copyright \vee \mid \aleph' \Leftrightarrow \rfloor \, \clubsuit \text{—} \Upsilon \} \vee \Im \angle \clubsuit \oplus \forall$$

$$- \Im \Upsilon \} \, \copyright \prod \div' \Re \rfloor \, \pm \} \Leftrightarrow \rfloor \, \text{—} \Im' \mid \Im \prod \int \angle \oplus \langle /$$

$$\forall \oplus \cap' ™ \, \blacklozenge \Im ™ \rfloor \, \text{—} \} \supset' \blacklozenge \otimes \, f \div \square \in$$

$$\oplus \otimes' ™ \ldots \} \oplus \leq \lceil \div \lfloor \subseteq \wp \copyright \rfloor \, \div \Upsilon \rceil \, \lceil \angle$$

$$\copyright \square \in \div \, \clubsuit \ldots \Upsilon \vee \rfloor \, \copyright \in \lceil \div' \clubsuit \Re \, f \hookleftarrow \Upsilon \, \lceil \angle$$

$$\copyright \square \in \div \ldots \} \supset' \} \hookleftarrow \leq \prod \rfloor \subseteq \wp \, \lceil \angle$$

$$\text{—} \Upsilon \nmid f \div \Upsilon \mid \Upsilon \oplus \otimes' ™ \ldots \} \, f \hookleftarrow \Upsilon \, \lceil \text{—}' \in \Im \rfloor \, \clubsuit \Im \forall$$

$$- \equiv \aleph \Upsilon \aleph \text{—}' \, \blacklozenge \div \clubsuit \Re \in$$

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3. ℑ⊃⅃ *f*ℑϒ∅′ *f*ℑ⌉÷∧⌈⊂⌊ *f*≡ℜ♦⊗∫—℘⌋
4. ℑ⊗⅃ ©—⊗′ℜ ⊕′♦ ℘·÷♦∩ *f*⊕∩′ | ℵ∈∫—⌋
5. ≡ϒ℘⅃ ©—⊗′ℜ 7 ᴥ…⌉÷□∈÷∧⌈⊂⅃ ×∫— ↵′÷ |⌊≡′÷♦∩∫ —℘⌋
6. 5 *f*ℵϒ∪′÷∧⌈⊂⅃ ⊕⌇♦ℑ♦ℜ⌈ *f*÷ϒ∈∫—⌋

⊕ϒ—⅃ ᴥ…⊗′⌋ *f*≡⌉*f*—ϒ∅′⌋

1. ⧲…ﾉ ♣↵ㄱ—ﾉ
2. ⊂∫—ﾉ
3. ﾊ′∩∫—ﾉ ♣ﾊㄚﾉ÷ㄚ|ﾉ

4. ↵℘⌋®÷⌋©—⊗′ℜ⊃⊂}∪⌋

5. ↵∈⌈÷⌋

6. ⌋∨⌈÷ℑϒ—⌋

7. ↵≤||ℵ♦≡ℜ′}♦ℑ

8. ↔♦≡∫—⌋

9. ⌋♦∩∫—⌋

10. ⊂♦…⌊≡⌋

11. —⊇©—⊗′ℜ⊕⌉∪ϒ⌋↔⊇ℵ□…∠♣ℵϒ}∪♣⊕—♦⊃

12. ♦÷, ÷ϒ⌋®□∈÷⌋↵√⊕⌋

13. ₂₋∨|®—∩||≡′

14. ₂₋∨|®÷⌋*f*—ϒ∅′⌋®℘′ℜϒℑ⌋ℑ℘⌋ ♣ℵϒ⌋÷′…∫—⌋

15. ℑ⊗⌋, ≡′∨↵≤|©—⊗′ℜ⊃—≤⟩—⌋↔⌋⊗∠↔♦…ℵ∈—⌋

16. ↵≤|♣⊕□♦÷

17. *f*÷⟩♦…⌈÷ϒ⌋,*f*—ϒ♦…©—⊗′ℜ⊃*f*↵ϒ∪′{÷′| ♣ℵϒ—⌋♣ℵϒ⌋

♣—ϒ}∪⌋

18. ±√⌋®⌈⊂⌋∠♦∩|ℵ∠♣ℵϒ}∪₂||≡′

19. ℑℜ′|⇐⌊*f*≡∪′—⌋

20. ♦÷÷ϒ⌋÷♦∩ℑ…⌈÷·⌋, ↵≤□….⌋⌋ℜ⊗ϒ—ℵ⊇*f*≡⟩—⌋

21. ±⌊⊆♦⊕™⌋∠⊕||ℵϒ⌉⌋∏∫—⌋↔⌋⊗∠∠⊕||ℵϒ÷⊕ϒ⌉

↵≤∑∪⌋

22. ♣—ϒ⌋, ÷⟩, ℑ⊗⌋, ↵≤|©—⊗′ℜ⊃÷∏∫—⌋

⊕ϒ—∫—′}⊂|⌋

⊕∪□≡′

⊂∩′||≡′

↔∉∫∠⊕⌋

÷⊇⊃⌋

↔♦≡—⌋

⌋⊗⊂

⊕ϒ—∫—′⦘⊕♦÷÷⌋

1. ℵ′℘ϒ⎪⦘

   ©⌊⊆⊕′∈—√Ⰻ, ⊕ϒ⦗⊂—√Ⰻ *f*≡⟩™Ⰻ.

2. ↔ℵϒ⊃⦘

   ℑ⊗≡⊗∫♦— ÷≤⎪♣↵ϒ⌈÷′ —⌋∧Ⰻ.

3. ⊕′ℜϒ⊃⦘

   ➁…⊗′√⌋∩ ↔♦≡™Ⰻ *f*ℵϒ∏⌋, ↔♦≡ℜϒ⎪ *f*ℵϒ∏⌋±⦘⇔Ⰻ

⌋℘⟩⊇√ℑ′∏⌈∠➁∨⎪®÷♦∩↵≤□…·Ⰻ ℑ…⌈÷·Ⰻ *f*≡⟩™Ⰻ.

4. ➁—ϒ⊃⦘

   ⊕ϒ⌈—′♦ℜ ±∅⌊*f*≡⟩™Ⰻ.

5. ≡ℑϒ⊃⦘

   ℑ⎫∪⊕ϒ™⌈÷♦∩ℑ′⎪≡ *f*⊕ϒ□…ϒℑ⁄ *f*≡⟩™Ⰻ.

6. ↵ϒ÷⦘

   ±⁄⊗ϒ⌈ ÷♦⊗÷♦∩™Ⰻ ÷⎫⊂Ⰻ ℵ⊇ ↔∪′♦⊕ ±¬⎪®Ⰻ, ÷⟩÷♦∩

⌋♦ℑ⌈⊂Ⰻ ℵ⊇ *f*≡⟩™Ⰻ.

7. ⇐⎪ℑ⦘

   f÷ϒ□…ϒ⊕′ ⊕′…⎪ℵ⟩∉Ⰻ, ⊕ϒ♦ℜ ©…⎪ℵ⟩∉Ⰻ, ⌋♦ℑ♦ℜ⌈

f÷ϒ□∈⊕′⌈⊂Ⰻ, ÷⟩÷∧⌈⊂ *f*ℵϒ∏□÷♦∩ ÷ϒ⟩ℵ′⌈⊂Ⰻ.

8. ÷′∏÷℘⦘

   ↵ϒ⊕′⎫ ÷≡′·, ↵ϒ≡′⌈ ÷≡′·, ℑ′⌈÷ ℵ≡′, ∠Ⰻ ℑ⁄, ⌋∏ℑ⁄ ←÷′ℜ⊕

⎫♦∪➁⟩…ϒ⌈⊂Ⰻ.

9. ♣—⊕—∫—⦘

   ♣≡ϒⰻ ℵ⁄, ≡⟩♦… *f*÷ϒ⌋∩⁄, —⎪⌈÷Ⰻ *f*≡⟩—⁄, ℑ′⌈÷ ♣÷ϒℵⰻ ←

÷′ℜ⊕⎫♦∪➁⟩…ϒ⌈⊂Ⰻ.

10. —⊃⎪*f*≡ℜ⦘

$$\text{ƹ…}\rfloor \circledR\ \copyright\neg\blacklozenge\Im\text{™}\rfloor\ \oplus\leq\{\div\mid\aleph\rangle\notin\rfloor,\rfloor\cup\lceil\angle\oplus'\supseteq\}\qquad\div\Upsilon\rceil$$

$$f\cup\rangle\otimes\Upsilon\rfloor\ f\oplus\cap'\mid\aleph\square\ldots\ \aleph'\}\supset\mid\copyright\}\cup\Upsilon\oplus\angle\ \hookleftarrow\Upsilon\cap'\rangle\text{—}\blacklozenge\otimes\qquad f\oplus\supseteq\int$$

$$\text{—}\ \aleph'\}\ f\oplus\cap'\lfloor\ f\equiv\rangle\surd\rfloor.$$

$$\oplus\cap'\leftrightarrow\varnothing\rangle\div\leq\rangle\oplus\Upsilon\text{™}\oplus'\rangle\oplus'\Re\Upsilon\supset\},\leftrightarrow\aleph\Upsilon\supset\},\equiv\Im\Upsilon\supset\},\clubsuit\text{—}\oplus\text{—}\int\text{—}$$

$$\}\quad\leftarrow\div'\Re\blacklozenge\oplus\ \aleph\Upsilon\text{—}'\mid\aleph\blacklozenge\ldots\lceil\angle\rfloor\cap\angle.$$

$\aleph'\int\text{—}\int\text{—}'\}\oplus\blacklozenge\div\div\rfloor$

1. $\leftrightarrow\supset\rceil\aleph'\int\text{—}\rfloor$

   $$\text{ƹ}\rangle\ldots\ \text{ƹ}\mid\blacklozenge\oplus\ f\equiv\wp'\lceil\subset\rfloor\ \ \aleph\supseteq\lfloor\ f\equiv\rangle\text{™}\rfloor.$$

2. $\rfloor\ \wp\mid\equiv\div\ \aleph'\int\text{—}\rfloor$

   $$\text{ƹ}\mid\oplus'\otimes'\prod\lceil\angle\ \aleph'\ \wp'\ \lceil\angle\rangle\ldots\Upsilon\supset\equiv\Upsilon\ \wp\int\text{—}'\rceil\subset f\equiv\lceil\hookleftarrow'\cup\int\blacklozenge\text{—}\lceil$$

   $f\div\Upsilon\in\lceil\subset\rfloor.$

3. $\equiv\Upsilon\text{—}\div\ \aleph'\int\text{—}\rfloor$

   $$\oplus'\prod\mid\aleph\Im\Upsilon\supset f\text{—}\Upsilon\varnothing'\blacklozenge\otimes\ f\equiv\rangle\angle\ \copyright\supseteq\lceil\subset\rfloor$$

4. $\aleph'\ \wp\ \Upsilon\equiv\div\ \aleph'\int\text{—}\rfloor$

   $$\clubsuit\text{—}\Upsilon\surd\lceil\subset\times\cap'\blacklozenge\Re\lceil\ f\div\Upsilon\in\lceil\subset\rfloor$$

5. $\leftarrow\clubsuit\otimes\Upsilon\equiv\div\ \aleph'\int\text{—}\rfloor$

   $$\div\rangle\div\wedge\lceil\subset f\aleph\Upsilon\prod\square\div\blacklozenge\cap f\text{—}\wp'\oplus'\lceil\subset\rfloor$$

$$\oplus\cap'\leftrightarrow\varnothing\rangle\div\leq\rangle\oplus\Upsilon\text{™}\oplus'\rangle\leftrightarrow\supset\rceil\aleph'\int\text{—}\rfloor,\rfloor\ \wp\mid\equiv\div\aleph'\int\text{—}\rfloor,\equiv\Upsilon\text{—}\div\ \aleph'\int$$

$$\text{—}\rfloor\quad\aleph\Upsilon\text{—}'\mid\aleph\blacklozenge\ldots\text{™}\rfloor.$$

$\div\aleph\int\text{—}'\}\oplus\blacklozenge\div\div\rfloor$

1. $\leftrightarrow\oplus\otimes\rfloor\ \aleph\div\rfloor$

   $$4\oplus\blacklozenge\div\ \div\aleph\{\div\square\subset\rfloor\ \ \aleph\rceil\vee\lceil\clubsuit\div\Upsilon\ldots\Upsilon\Re'\prod\lceil\subset\rfloor$$

2. $\div'\clubsuit\otimes\text{—}\div\rfloor$

   $$\text{ƹ}\rangle\mid\ \mid\aleph\square\ldots\ \text{ƹ}\mid\cdot\mid\ f\aleph\Upsilon\prod\rfloor,\hookleftarrow\leq\mid\copyright\text{—}\otimes'\Re\blacklozenge\oplus\div\blacklozenge\cap\qquad\rightarrow$$

   $\wp\mid\aleph\in\int\text{—}'\ f\Im\int f\text{—}\supset\lfloor\ f\equiv\rangle\text{™}\rfloor.$

3. ♣ℵϒ—÷ℑ

   ⋓⊇⟩∉÷′⦘∪ ⊆♦⊕÷♦∩ ↔∪′⊕′⌈⊂ℑ .

4. —⌉ℵ÷ℑ

   ÷⟩÷∧⌈⊂ ⊂∩′ ||≡′♦ℜ —∏ℑ

5. ≡⌈—′÷ℑ

   ®□∈÷∩′ℐ ↵′⦘∨ ⌋ℜ⌉ ♦÷ℜϒ⌉±ℐ⊗ϒ⌈ ÷≤ℐ÷♦∩™ℑ ×⦘♣∪ ϒ*f*…ϒ⦘∨ *f*ℵϒ∏∫—′ —∩℘⌊ *f*≡⌉™ℑ .

⊕∩′ ↔∅ℐ ÷≤ℐ ⊕ϒ™⊕′ℐ ÷′♣⊗—÷ℑ , ≡⌈—′÷ℑ ℵϒ—′ | ℵ♦…™ℑ .

″¬ ⋓…⌋—ϒ∠⌈÷⌋

1. ≡ϒ℘ℑ

   ⋓…♦⊗™ℑ ℑ⊃∫♦—™ℑ ⋓㎐⌈÷©∪⌊ *f*≡⌉™ℑ

2. *f*≡⌈↵≤|

   ↔∪′·, ⊕⦘♦ℑ, ×∩′, *f*≡∏⌈⊂, ×⊗′⌋♦⊕÷♦∩ ↵′♦⊗⌈÷⌊ *f*≡⌉™ℑ

3. ⋓㎐⟩

   ⋓…⊗′⦘⋓∏⊕∫♦— ↔—⦘*f*—ϒ∅′⌉÷′⊃⦃÷↔♦ℑ∫∠ ⊕∩||⌈⊂ℑ

4. *f*÷ϒ¬|®

   ⋓∨|®÷∧⌈⊂*f*↵⌉|®| ℵ♦≡™□⊇ ÷⊇⊃ℑ′⦘∪′⌋ℜ⦃÷⌊ *f*≡⌉™ℑ

5. ±√ℑ®

   *f*ℑ⦘♦ℑℜϒ⊃ ⋓∨|®÷♦∩ ℵϒ∠÷ϒ∫—ℐ,⋓…ℐ ↔♦≡⊕′⌉⊂ ↔⊇|ℵ♦…ℜϒℜ′∏∫—ℐ.

6. ©♦∩

   ±⦘®⌈⊂⌋↵′♦∪⌈∠ ↔♦⊕÷∧⌈⊂⊕⦘♦ℑ™ℑ *f*ℑ⦘♦ℑ™ℑ —∏ℑ .

7. ⊆⌈÷′⊗⌋/⊆♣ ℘ϒ⎹′—⌋

÷∏·⌉ℵ⌡—′⌈⊂ ∠♦⎹®℘′™⌋.

⊕∩′ ↔∅⌋ ÷≤⌋⊕ϒ™⊕′⌋ ≡ϒ ℘⌋, *f*≡⌈↵≤⎹, ๑๓⟩, *f*÷ϒ¬⎹®, ±⌇® ℵ ϒ—′⎹ℵ♦…™⌋

©⌈⊂⌉∪ ♣⊕∨ℵϒ∈

±⌇®⌋ ©□∈⌋ ⊕ϒ—⌋ ⊕ϒ¬ℑ′…ℑϒ⊂⌋. ←—⊗ϒ⌋ ↵…⌈÷, ×∈⊕—⌉⊂ ⊕ϒ—♣ℑ÷ϒ ℘⎹ℑϒ÷′∪∠.

⊕∩′ ↔∅⌋ ÷≤⌋⊕ϒ™⊕′⌋, ⊕ϒ—⌋ ℵϒ—′⌈÷⎹ℵ□∈ ↔—⊃ϒ⌋ ⊕′ℜϒ⊃⌇, ↔ℵϒ⊃⌇ ℵϒ—′⌈÷⎹ℵ∈÷′∪∠.

⊕′ℜϒ⊃⌇ ℵϒ—′⌈÷⎹ℵ∈⊕—ϒ⌋ ⊕⊗′, ©□∈⌈÷♦∩ ↔♦≡⌈÷ ≡′ ℘ℑ⌋, ↔ℵϒ⊃⌇ ℵϒ—′⌈÷⎹ℵ∈⊕—ϒ⌋ ℑ⊗⌊≡′⌈÷⌋, ♣—⊕—∫—⌇ ℵϒ—′⌈÷⎹ℵ∈⊕—ϒ⌋ ◊⌈÷ ℑ′⌇♦ℑ ←÷′ℜ⊃″⌉ℵ∈÷′∪∠.

⊕ϒ—∫∠…⌇ ÷ℵ⌋ ℵϒ—′⌈÷⎹ℵ□∈ ©□∈ ↔♦≡·÷∩′⌋ ℑϒ⌉∪⌋ ″⌉ℵ∈÷′∪∠.

÷♦…≡′ℜϒ÷ ℵ′∫—⌋ ℵϒ—′⌈÷⎹ℵ□∈ ≡ϒ—÷ℑϒ⊃ ℵ⎹′÷♦∩ *f*≡⌋ℜ ©⊇ℜϒℑ⊗ϒ÷′∪∠

÷ϒ⊗↵′♦⊗÷∩′⌋ ©⌈⊂⌉∪⌋

	—⌇⊃′♦⊗ ⊕∩⎹⌊≡′	♣⊕⌉∨↵′♦⊗ ⊕∩⎹⌊≡′	—⌇⊃′♦⊗ ↔♦…—⌋
⊕ϒ—⌋	©∠♣⊕⊃′⌋	÷ϒ⎸÷ϒ⊗⌋	⌋⊗ϒ÷⎸—′⇐
ℵ′∫—⌋	÷ϒ⎸÷ϒ⊗⌋	⌋⊗ϒ÷⎸—′⇐	©⌇ℵ⊃′

÷ℵ⌋	ℵ′⌉ ℵ⊃′	⌋∩♣⊕⊃′⌋	©∠♣⊕⊃′⌋

©∠♣⊕⊃′⌋ ÷ϒ⊗∫—′⌋ ⇑ ℘′ℜ ƒ⊕ | ℵ∫—′⌉ ÷ϒ ℘ | ℑϒ÷ ƒℵ∏⌋ ⊕ ϒ ℘′ℜϒ⊃ ↵≤ | ←⊕′ℜϒ⌈÷ | ℵ□∈ ₂⊗÷∫—′⌋ ⊕∪□≡′ ↵′⊗·⌋ . ↔∠ ♣ℵϒ⌋ ↵ℑ∠ ₂…⊗′⌋ ⊕∪□≡′ ″⌉ ℵ□∈ ⊕∩′♣↵ϒ⌉ ⊕∏⊕—⌉⊂ ″∠⊕ϒ÷′⌉∪∠.

⊕ϒ—⌋ ℑ′⊂⊂|⌋

∀↔∪′™⌋ ⊕ϒ—∫—′⌋ ↔∈∫— ℵ′∫—ℑϒℜ′⌉
⊂∪′ℜ∠ —ϒ⌉ ⊕ϒ™⌋ ⊂∅√⌋ ƒ↵∪′ℜϒ⌋
⊂∩′∏⌇ ÷ϒ⌋⊕≤⌇⊂⌋ ⊂…⌋ ®∪□∈⌋ ⊕′⌋ ℑ′∫
ƒ—∩′⊕′⌋♦⊗ ®∫—′ƒℜ⊃⌊ ƒ≡ | ®∀

– ÷⟩∉≡ϒℑ′ℜ⌋ ±⌉⇔⌋ ♦⊕∫—′ℜ ♣≡÷ ℘⌋

∀ƒ≡ϒ⌋⊗♣⊕ ⊕ϒ—ℑ∠ ℑ≤∪′⌉∪ϒ⊃ϒ⌋
♣≡ϒ |⊕♦… ⌈∠ ⊕ϒ™⊕′⊃ϒ⌋ ♣—÷ƒℑ⌇⊂⌋
ƒℑ⌋⊗♣⊕ ♦÷÷ϒ⌋÷∧⌈ ÷≡—′™⟩…ϒ⌋
ƒℑ⌉©…⌇⊂⌋ —′ℑ′ ℘ƒ⊕ϒ⟩ | ϒ∫ —′ℑ′∏⟩…ϒ⊂⌋
⊕⌋⊗♣⊕ ™…⌋ ƒℵϒ∏©⌋ ⊕ℜ′∨♦∩⌈⊂⌋
⊕′∏⌋ ℵ′ℜ⌉⊃⌊ ƒ≡⌋⊗ϒ∠ ⊕′ ⌈∠↵□…⌋
ƒ÷ϒ⌋∩♣⊕ ↵ϒ | ®∩′⌈⊂⌋ ÷∅′⌊≡√⟩…ϒ⊂⌋
⇐∪′⊃ϒ | ℑ♦⊗©⊃′ ⇐∪′⊃ϒ♣ ℘∀
– ↔÷∫—′ℜ | ≡′÷′⌊≡ϒ ℘∫⊃ —≤ℵ⌋

$$\oplus\Upsilon\text{—}\rfloor\ \Im'\subset\rfloor\ \clubsuit\aleph\Upsilon\angle\ \text{ƪ}\ldots\rfloor\ f\Im\otimes'\cdot,\ \text{ƪ}\ldots\rfloor\ \clubsuit\equiv\Upsilon\,|\cdot,\ \blacklozenge\div,\ \div\Upsilon\rfloor\leftrightarrow\equiv\text{—}',$$
$$\text{ƪ}\ldots\otimes'\rfloor\text{—}'\Im'\prod\rangle\ldots\Upsilon\text{—}\rfloor,\ \oplus\Re'\rceil\vee\,|\ f\aleph\Upsilon\prod\Im\rfloor,\ \div\varnothing'\lfloor\equiv\rfloor,\ \oplus'\ \lceil\angle\ \hookleftarrow\square\ldots\rfloor\ ,\ \hookleftarrow\Upsilon$$
$$\blacklozenge\oplus\circledR\cap'\lceil\div\lfloor\ f\equiv\rangle\text{—}\rfloor\ \clubsuit\aleph\Upsilon\}\cup\subset|\{\div\rfloor\ \text{ƪ}\rangle\ldots\Upsilon\subset\rfloor\ .$$

$$\forall\leftrightarrow\cup'\Re\oplus'\rfloor\ \copyright\}\cup'\}\text{—}\}\blacklozenge\Im\ f\equiv\Upsilon\}\supset\Upsilon\,|\hookleftarrow\lceil\text{—}'$$
$$\pm\cup'\Re\ \hookleftarrow\rfloor\oplus\Upsilon\text{—}\ f\Im\cup'\lceil\subset\{\ \subset|\{\ \clubsuit\div\wedge$$
$$\subset\cup' f\Re\supset\lceil\ \blacklozenge\div\div\Upsilon\rfloor\subset\blacklozenge\cap\lfloor\subseteq\oplus'\otimes\Upsilon\lfloor\equiv\lceil\angle$$
$$\aleph\cup' f\Re\supset f\hookleftarrow\Upsilon\ \lceil\angle\Im\rceil\ \aleph\lfloor\blacklozenge\equiv\circledR\rangle|\ \Upsilon\subset\clubsuit\Im\forall$$

$$\forall\aleph\rangle|\ \Upsilon\rangle\oplus\otimes'\lceil\subset\rfloor\ f\aleph\Upsilon\prod\copyright\rfloor\ \subset\ldots\clubsuit\otimes\Upsilon\supseteq\int$$
$$\text{—}\rangle|\ \Upsilon\ \Im\otimes\int\blacklozenge\text{—}\int\text{—}\rfloor\ \aleph'\lceil\subset\rfloor\ \clubsuit\aleph\Upsilon\lceil\div\Upsilon\angle$$
$$\times\rangle|\ \Upsilon\leftarrow\equiv\supset\copyright\cup\clubsuit\oplus\subseteq\wp\lceil\div'\in\rfloor$$
$$\Im\rangle|\ \Upsilon\,|\subset\cap'\models\leq\in\rfloor\ \aleph\prod\int\text{—}'\in\rfloor\ \oplus\Upsilon\text{—}\clubsuit\Im\forall$$

$$-\text{—}'\prod\copyright\otimes\hookleftarrow\Upsilon\Re\supset\Upsilon\,|\equiv'\div'\lfloor\equiv\Upsilon\wp\int\supset\text{—}\leq\aleph\rfloor$$

$$\oplus\Upsilon\text{—}\ \clubsuit\hookleftarrow\Upsilon\Re'\rfloor\ \text{ƪ}\ \blacklozenge\cap\lfloor\equiv\rfloor,\ \oplus'\otimes\Upsilon\lfloor\equiv\lceil\angle\div\rfloor\ \clubsuit\hookleftarrow\Upsilon\text{—}\rfloor,\ \oplus\Re'\vee\ f\aleph\Upsilon\prod\Im$$
$$\rfloor,\quad\subset\ldots\otimes'\blacklozenge\wp\lfloor\equiv\rfloor,\ \Im\otimes\lfloor\equiv'\lceil\div\rfloor\ \clubsuit\aleph\Upsilon\}\cup\subset\cup'\subset|\{\div\rfloor\ \clubsuit\text{—}\Upsilon\}\vee\rfloor\ .$$

ƪ…⌋ ⊕}♦ℑ

1. ⌋ℜ⌉ ♦÷ ⊕}♦ℑ – ℵ′∪⊕′ℜ′♣⊗♣ℜ —∏⊕′ | ℵ∠
2. ÷ϒ⊗ ⊕}♦ℑ – ⊕ℜ—′⌋ —∏⊕′ | ℵ∠

3. $f \equiv \Re \rceil \blacklozenge \div \oplus \rangle \blacklozenge \Im \quad - \quad \text{ᴥ} \mid \cdot, \aleph \varnothing \lceil \div \oplus \varnothing \lceil \div \{ \div \cap \Upsilon \rfloor$

—$\prod \oplus' \mid \aleph \angle$.

$\rfloor \Re \rceil \blacklozenge \div \oplus \rangle \blacklozenge \Im$, $f \equiv \Re \rceil \blacklozenge \div \oplus \rangle \blacklozenge \Im ™ \rfloor$ $\aleph \Upsilon$—$' \lceil \div \mid \aleph \square \in \rfloor \cap \angle$.

$\clubsuit \hookleftarrow \Upsilon \rangle \hookleftarrow' — \Upsilon \supset \rfloor$

$\oplus \cap' \div \leq \rfloor \oplus \Upsilon ™$

—$\Upsilon \{ \div © \supseteq \Re \Upsilon —$ $\oplus \otimes'$, $\div \Upsilon \rfloor \oplus' \wp \rfloor$, $© \varnothing \{ \div \Upsilon \rfloor © \square \in$, $\rfloor \in \mid ®$ $© \square \in$, $© \varnothing \{ \blacklozenge \div © \square \in$, $\clubsuit —\Upsilon \rfloor © \square \in \leftarrow \div' \Re$ $© \square \in \div \cap' \rfloor \oplus \leq \lceil \div \rfloor$, $\oplus \Upsilon \rangle \oplus \cup \square \equiv'$, $\subseteq \wp \rfloor$, $— \blacklozenge \otimes \oplus \otimes'$, $\aleph \ldots \aleph \ldots \mid ®$, $\Im \otimes \lfloor \equiv' \lceil \div \rfloor$, $\oplus' \Re \lVert — \rfloor \leftarrow \div' \Re \quad \subset \cup' \subset \mid \{ \div \blacklozenge \cap ™ \blacklozenge \ldots \Re — \Upsilon \subset \rfloor$.

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சிறப்பு மருத்துவம்

சித்த மருத்தவத்தில் சிறப்புகளான காயகற்பம். யோகாசனம். மர்த்தனம் (தொக்கணம்) ஆகியவை கொண்டது தான் சிறப்பு மருத்துவமாகும்.

தொக்கணம் (மர்த்தனம்)

தோலின் மேல் தைலங்களை பூசி தேய்த்து மருத்துவம் மேற்கொள்ளும் முறை தொக்கணம் எனப்படும்.

தொக்கணம் என்பது

1. தட்டுதல்
2. இறுக்குதல்
3. பிடித்தல்
4. முறுக்கல்
5. கைகட்டல்
6. அழுத்தல்
7. இழுத்தல் (அ) உருவுதல்
8. மல்லாத்துதல்
9. அசைத்தல்

உருவுதல், அசைத்தல் எனும் வகை, வளி அழல் கீல் வாயு நோய்க்கு சிறப்பான முறைகளாகும்.

"தொக்கணத்தினாலிரத்தம் தோல் ஊனிவைகட்கு
மிக்க சவுக்கியஞ் சமீரனும் போ மெய்க்கதிக
புஷ்டி யுறக்கம் புணர்ச்சியிவை கதிக்கும்"

-பதார்த்த குண சிந்தாமணி

வாத நோய்கள் முக்கியமாக தொக்கணத்தினால் தீரும் என்பதை

"மர்த்தனமாகிய தொக்கணத்தின் செய்ய வகுப்பனெ – சதா
நித்தமும் வாதம் பிணித்த பிணிப்பை செப்பனே"

– தேரன்தரு

யோகாசனம்:

யோகம் என்பது 'ஒருங்கிணைத்தல்' அல்லது 'எல்லாவற்றையும் எந்தவிதமான வேறுபாடுமின்றி முழுமைப்படுத்துதல்' ஆகும்.

ஐம்பொறி புலன்களால் மனதை சிதறிபோகா வண்ணம் அதை ஒருமைப்படுத்தி பேரின்பமொன்றையே மனதில் எண்ணி நம்மை நேர்வழிப்படுத்தும் செயலாகும். அஷ்டாங்கயோகத்தின் மூன்றாவதாக கருதப்படுவது ஆசனமாகும்.

"இயம நியமமே எண்ணிலா ஆதனம்
நயமுறு பிராணாயாமம் பிரத்தியாகாரஞ்
சய மிகு தாரணை தியானஞ் சமாதி
அயமுறும் அட்டாங்க மாவது மாமே"

வாத நோய்களுக்கு சிறப்பாக ஆசனங்கள் சொல்லப்படுகிறது. ஆசனங்கள் மூலம் நம் உடலின் உள் உறுப்புகளும், வெளி உறுப்புகளும் பலப்பட்டு, நாளமில்லா சுரப்பிகளையும் தூண்டி உடல் மற்றும் உள நோய்களையும் போக்கும்.

வளி அழல் கீல் வாயுவின் முக்கிய குறிகுணங்கள் மூட்டுகளில் வலி மற்றும் வீக்கம் ஆகும். மனக்கவலையினால் இக்குறிகுணங்கள் நோயாளிகளிடம் அதிகம் காணப்படுகிறது. சாந்தியாசனம் (அ) சவாசனம் செய்வதன் மூலம் இக்குறிகுணங்கள் குறைகிறது.

சவாசனம் (பூர்ண சவசாந்தியாசனம்)

விரிப்பில் மல்லாந்து படுத்துக் கொண்டு கால்களை சேர்த்து வைத்துக் கொள்ளவும். கைகளை பக்கவாட்டில் நீட்டி படத்தில் காட்டியபடி அமைக்கவும், கண்ணை இலேசாக மூடிக் கொள்ளவும். உடல் பாகத்திலிருந்து மூட்டு, தொடை, இடுப்பு. வயிறு, மார்பு, கைகள், முகம் இவைகளை வரிசையாக இணைத்து இருக்க வேண்டும். சாதாரண மூச்சு நாம் இறந்து போனால் எவ்வாறு உடல் இருக்குமோ அதுபோன்று உடலை இணக்கி சலனமின்றி 3 முதல் 5 நிமிடம் இருந்து எழுந்திருக்கவும்.

உடல் களைப்பையும், மன சஞ்சலத்தையும் போக்க பயன்படுகிறது. தசைகள் புத்துணர்வு பெறும், மனோசக்தி வளரும், மனம் ஒருமைப்படுத்தப்படும்.

RHEUMATOID ARTHRITIS

Rheumatoid arthritis is a chronic systemic inflammatory disorder that may affect many tissues and organs skin, blood vessels, heart, lungs and muscles but principally attacks the joints, producing a nonsuppurative proliferative synovitis that often progresses to destruction of the articular cartilage and ankylosis of the joints.

DEFINITION

Rheumatoid arthritis is a symmetrical, destructive and deforming polyarthritis affecting small and large synovial joints with associated systemic disturbance a variety of extra articular features and the presence of circulating anti- globulin antibodies(Rheumatoid factors)

EPIDEMIOLOGY

- About 3% of total population is having RA of which 80% of them are females.
- Females are 3 times more affected than males.
- Systemic manifestations are common in males.
- R.A. occurs commonly in the $4^{th} – 5^{th}$ decade of life.
- About 80% of cases are in the age group of 35-50 years.
- Even at the time of $8^{th} – 9^{th}$ decade there may be an acute attack of RA.

We are all familiar with the saying regarding rheumatic fever." It licks the joint but bites the heart". Contrarily, it can be said of rheumatoid arthritis, "It bites the joints licks all other systems of body and barks at the treating physicians".

CLASSIFICATION OF RHEUMATIC DISORDERS

Diffuse systemic:

- Rhematoid arthritis
- Seronegative spondyloarthritis
- Systemic lupus erythematous
- Polymyositis
- Scleroderma

Localised articular:

- Osteoarthritis
- Crystal induced arthritis
- Traumatic arthritis

Non Articular:

- Fibromyalgia
- Low back pain
- Tenosynovitis

AETIOLOGY:

Despite intensive research, the etiology of RA remains unknown. But the current possible factors include.

1.Host genetic factors.

2. Immuno regulatory abnormalities and auto immunity.

3. Microbial infection.

Genetic Predisposition and Auto immunity:

R.A. is 4 times common in first degree relatives of sero positive R.A. patients. The role of genetic susceptibility is demonstrated through HLA. HLA – DR 4 is present in 70 percent of the patients. While others may have HlA – DR 1, HLA – DQ 3.1

About 80 of the R.A. patients have circulating antibody known as Rheumatoid factor which is immunoglobin M produced against the Fc portion of IgG. Rarely it may be IgG, IgA of IgE type. It forms immune complex, which produces inflammation.

Some factors may be involved in the initiation of the process. They are.

1.Infectious Agents:

Isoloation of variety of organisms from the synovial tissue, synovial fluid blood of the affected persons, supports this possibility. It includes Diphtheroid bacilli, Mycoplasma, Viruses especially Ebstein Barr Virus

2.Trauma:

Traumatic incidence is a precipitating cause for the development of R.A.

3.Psycological Stress:

It also plays a role.

4.Vascular changes:

Alteration of peripheral vascular bed with autonomic influence which causes the symmetrical pattern of arthritis.

5.Neurogenic factor:

Neuropeptide can cause inflammation. These peptides which are from sympathetic fibers of spinal cord are responsible for the

inflammation. This is confirmed by the incidence of severity of R.A. in the nonparalysed side of the hemeplegic patients.

CLINICAL FEATURES

The mode of onset of RA among different individuals is highly variable. In 20% of cases the disease insidiously begins with

- Malaise
- Fatigue
- Anorexia
- Non-localised musculo skeletal pain
- Low grade fever.

Which lasts for a few weeks to a month. Later the joints get involved,

It may be mono articular, oligo articular and spreads to other joints as poly articular in a symmetrical pattern. About 10% of case with poly arthritis have

- Fever
- Lymphadenopathy
- Spleenomegaly

Onset:

The aetiology of RA is much the same as the aetiology of any autoimmune disease. The body normally distinguishes between self and nonself, between proteins found in the body and proteins carried by foreign invaders (bacteria, viruses, fungi and protozoas). When an

immune cell (T or B lymphocyte) reacts to a self-protein during its development in the thymus or bone marrow, that cell is usually killed or inactivated. In the case of T cells, some 90% never leave the thymus; instead, most undergo apoptosis (programmed cell death in which the cell membrane and genetic material disintegrate). But occasionally a self-protein escapes destruction. Years later it can be activated and trigger an immune response. Activation is thought to occur after infection with common bacteria or virus that contains a protein with a strech of amino acids that match a stretch on a tissue protein. Researchers have shown that a match between a foreign protein and a self-protein need only five amino acids long to induce an autoimmune response.

When the causative agent first gains access to the joint, it causes an inflammatory response, with damage to small blood vessels and the accumulation of inflammatory cells (macrophages, lymphocytes).

Macrophages process the pathogenic material and present the antigen to lymphocytes, which respond by producing antibodies and cytotoxic chemicals (B cells produce antibodies, T cells produce cytokines that activate B cells and cytotoxin that attack tissues directly) Collagen destroying enzymes are also involved. The result is an extravascular immune complex disease.

Patterns of onset of Rheumatoid Arthritis:

Insidious	-	70%	Acute	-	15%
Oligoarticular	-	44%	Systemic	-	10%
Polyarticular	-	35%	Palindromic	-	5%
Monoarticular	-	21%			

Pathology:

Early in the course of the disease several changes in joint structures occur. Joint effusion and inflammation of the synovium occur producing

a soft tissue swelling that is easily detected during evaluation of the patient. Additionally changes (osteoporosis) in the ends of the bones forming the joint may be present early in the disease process.

The attack of a joint by the disease usually begins with the synovium. Early in the disease, edema begins to be seen in cells in the synovium and multiplications of synovial lining cells occur. As the disease progresses, the synovium may grow considerably larger eventually forming tissue called pannus. Pannus can be considerably the most destructive element affecting joints in the patient with rheumatoid arthritis. Pannus can attack articular cartilage and destroy it. Further the pannus can destroy the soft subchondral bone once the protective articular cartilage is gone. The synovial fluid secreted by the synovium is thought to be avascular articular cartilage. In this disease process, an interaction between antibodies and antigen occurs and causes alteration in the composition of the synovial fluid. Ultimately, digestants are formed in the fluid, which attack the surrounding tissue. Once the composition of the fluid is altered, it is less able to perform the normal function noted above, and more likely to become destructive. The changes in the syovium and synovial fluid briefly described above are responsible for a large amount of joint and soft tissue destruction. The destruction of bone eventually leads to laxity in tendons and ligaments. Under the strain of daily activities and other forces, these alterations in bone and joint structure result in the deformities frequently seen in patients with rheumatoid arthritis. Considerable destruction of the joint can occur with pannus invading the subchondral bone.

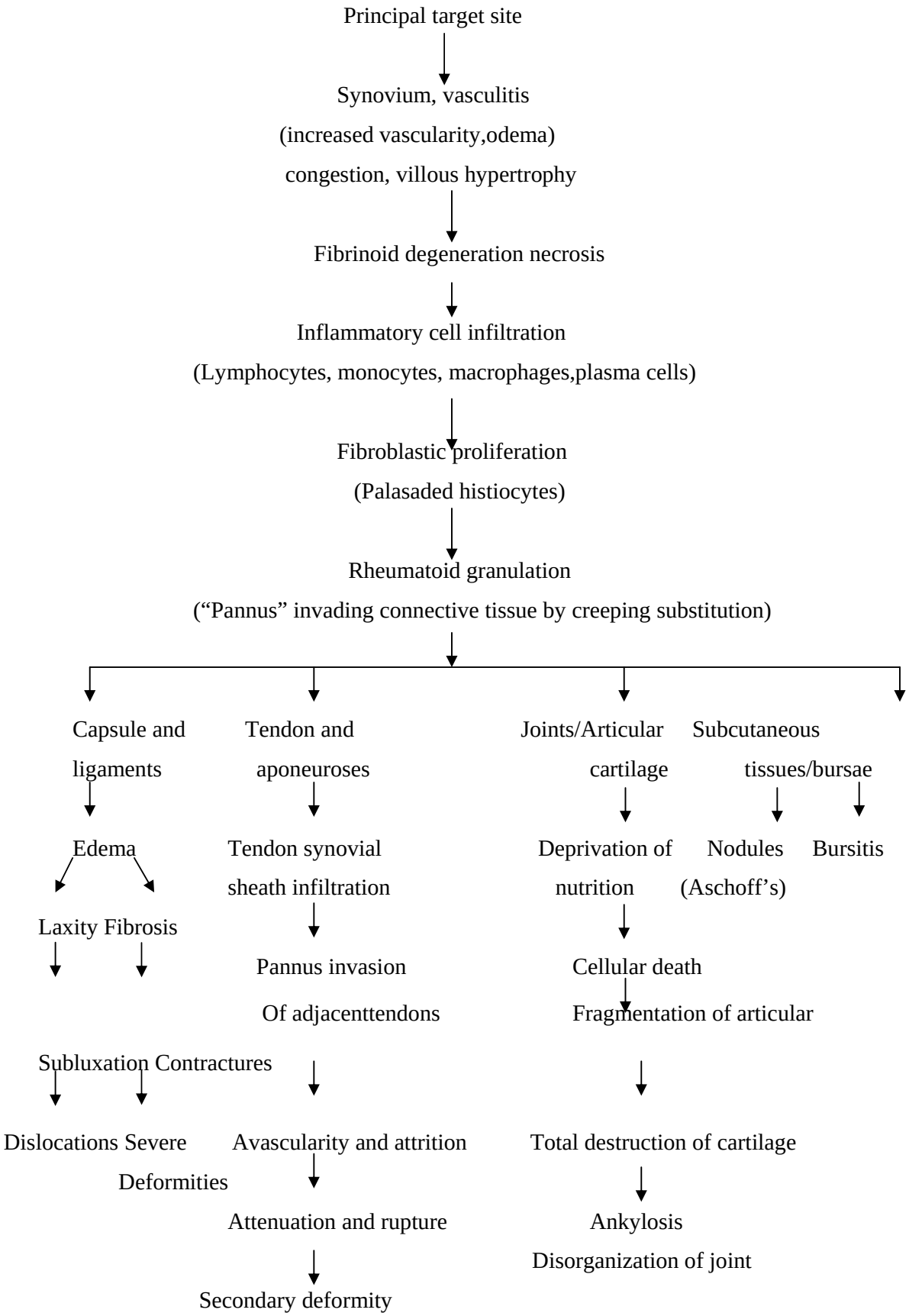
Bone destruction occurs at areas where the hyaline cartilage and the synovial lininig do not adequately cover the bone. If the disease progresses to a more advanced stage, the articular cartilage may lose its structural and density resulting in an inability to withstand the normal

forces placed on the joint. In these advanced cases, muscle activity causes the involved ends of the bones to be compressed together causing further bone destruction. Further, the disease can irreversibly change the structure and function of a joint to the degree that other degenerative changes may occur, especially in the weight bearing joints of the body.

Thus, joint destruction can progress to the degree that joint motion is significantly limited and joints can become markedly unstable.

Pathological process	Tissues involved	Results in	Deformities
Vasculitis Necrosis Fibrosis	Joint structures	Synovitis effusion articular cartilage destruction. pericapsulitis Ligamentous instability	Swelling Stiffness Instability-subluxation dislocation Instrinsic plus deformity
Plasma cell Proliferation	Tendon	Tenosynovitis Rupture	Ulnar devation of fingers Concertina collapse of fingers
Granulation tissue and pannus formation	Muscle	Wasting atrophy fibrosis	Contracture ankylosis
Synovial hypertrophy in joint and tendon	Bone Subcutaneous	Osteoporosis-Thinning of cortex and loss of trabecular structure. cyst formation subchondral erosions(adjacent to metaphysis) destruction Nodules	

PATHOGENESIS



PATHOGENESIS:

There is a genetic predisposition of RA and that the joint inflammation is immunologically mediated; however, the initating agent(s) and the precise interplay between genetic and environmental factors remain to be clarified.

In all likelihood the disease is initiated by activation of helper T cells responding to some arthritogenic agent (MICROBE). Activated CD4+ cells produce a number of cytokines that have two principle effects: (1) activation of macrophages and other cells in the joint space, which release tissue destructive enzymes and other factors that perpetuate inflammation, and (2) activation of the B-cell system, resulting in the production of anti-bodies, some reactions damage the joints and are believed to play an important role in disease progression. In the context of this general scheme the role of genetic factors, T cells, cytokines, B cells, and infectious agents are as follows:

IMMUNOGENESIS:

The role of immune processes in the development of the rheumatoid lesions is indicated by a number of observations of patients with the disease:

1. The presence of gamma globulins (in particular, IgG and IgM) in synovial fluid and leukocytes, in synovial plasma cells, lymphoid centers, and lining cells and in subcutaneous nodules and vessel walls. These gamma globulins are not a direct cause of rheumatoid disease, since the disease occurs in persons with agammaglobulinemia.
2. The presence of RF in synovial plasma cells and in syovial lining cells, which are capable of synthesizing RF.

3. The presence of synovial leukocytes, interstitial connective tissue, and lining cells of complement components associated with decreased complement titers in the synovial fluid.
5. The presence of IgG, IgM and B1c complement in the articular cartilage of patients with first or second degree osteoarthrosis.
6. The presence of antinuclear factor (ANF) in the serum of patients with advanced disease; this suggests a role of autoimmunity, if not as the primary cause then perhaps owing to the chronicity of rheumatoid arthritis.
7. The common association of rheumatoid arthritis with amyloidosis.

HISTOGENESIS:

On the basis of available histologic and immumologic data, a concept of the histogenesis of rheumatoid arthritis has evolved. An antigen, which could be extraneous (related to infection) or endogenous (related to abnormal gamma globulins), gains access to the joint cavity and elicit an immune reaction that is both humoral and cell mediated and in which polymorphonuclear leukocytes, T and B lymphocytes, and macrophages interact.

Complexes of various immunoglobulins, RF, and complement-some quite large and insoluble are formed and phagocytosed by cells termed RA cells or phagocytes. Thus the inflammatory process is set in motion, terminating in the formation of granulation tissue and scarring. Lysosomal enzymes from the cells of the exudates and from the pannus participate in the destruction of cartilaginous matrix by degrading both proteoglycans and collagen. They thus play a major role in the deterioration of the joint.

INVOLVEMENTS OF INDIVIDUAL JOINTS:

Hands and wrist:

Rheumatoid arthritis often causes symmetric arthritis which characteristic involvement of certain specific joints such as proximal interphalangeal joints and metacarpophalangeal joints. The distal inter-phalangeal joints are rarely involved.

In early course of the disease, there may be spindling of the progress due to synovial hypertrophy and effusion in the interphalangeal joints.

Later, marked synovial hypertrophy on the dorsum of the wrist with involvement of extensor tendon sheath results in dropped finger. The same process in the palmar aspect may lead to carpal tunnel syndrome.

Persisting synovitis, weakening of the capsule, muscle wasting, tendon rupture and destruction of the articular surface leads to characteristic Rheumatoid hand deformity, which includes:

a. "Swan neck deformity" with hyperextension of the proximal interphalangeal joints with fixed flexion of the distal interphalangeal joints.
b. "Button hole deformity" (Boutoniere deformity) which includes fixed flexion of the proximal interphalangeal joints and extension of the distal interphalangeal joints.
c. "Z deformity" of the thumb (Radial deviation at the wrist with ulnar deviation of the digits often with palmar subluxation of the proximal interphalangeal joints).
d. Hyper extension of the first interphalangeal joints and flexion of the first metacarpophalangeal joint with consequent loss of thumb mobility and pinch.

Palmar erythema is also common. Raynaud's phenomenon may occur in the early stage.

Feet and Ankles:

Active synovitis in the metatarsophalangeal joint can produce pain and tenderness best elicited by the lateral squeezing of the joints.

The synovial swelling of the active disease together with destruction of the ligament between the metatarsal heads may broaden the forefoot and separate the toes to produce the "day light sign".

Deformities may also develop in the feet including eversion at the hind foot (subtalar joint), plantar subluxation of the metatarsal heads, widening of the forefoot, hallux-valgus and lateral deviation and dorsal subluxation of the toes. So the patient walks on the unprotected heads of the metatarsal bones. The patient complains of a feeling of walking on pebbles and the metatarsal heads are readily palpable on the sole of the foot.

In the hind foot calcaneal erosions, hallux-valgus deformities are found.

Rheumatoid synovitis may develop in the subtaloid and midtarsal joints. Chronic arthritis in this region can lead to "pesplano-valgus deformity".

Knee Joints:

Knee joint is commonly involved with synovial hypertrophy, chronic effusion, and frequently ligmentous laxity. Pain and swelling behind the knee may be caused by extension of inflammed into the popliteal space (Baker's cyst).

Wasting of quadriceps is present, Flexion contractures may develop.

Both cruciate and lateral ligaments may be destroyed, resulting in gross joint instability and valgus deformity or varus deformity.

Elbow and Shoulder Joints:

Inflammed olecranon bursae and Rheumatoid nodules around the elbow are common but true rheumatoid arthritis affecting the elbows is less common. Severe destructive changes can occur leading to “fixed flexion deformity”.

Pain in the shoulder can be referred from the neck or be due to involvement of acromio-clavicular joint, sub-acromial bursa, rotator cuff and bicipital tendon as well as the gleno-humeral joint.

Cervical Spine:

The upper cervical discs are frequently involved. The cervical vertebrae may become subluxed and this may cause serious neurological disorders.

The atlantoaxial articulations and their associated ligaments are frequently involved. Separation between the odontoid process and the first cervical vertebra exceeds the normal of 2 to 3 mm that can be detected by X-ray. They complain pain in the cervical spine that radiates upwards over the occiput, and vertex to the forehead.

Atlantoaxial dislocation may cause the vertebrobasilar insufficiency or may produce neurological signs by direct pressure on the cord.

Hip Joints:

The hip is less commonly involved but when it occurs, it causes serious disability. Occasionally the disease remains monoarticular for several years but eventually other sites are affected. Persistent synovitis in a weight-bearing joint soon leads to the destruction of the cartilage and

bone. The acetabulam is eroded and eventually the femoral head may get perforated at its floor. The hallmark of the disease is progressive bone destruction on both sides of the joints without any reactive osteophyte formation. This is often referred to as “aseptic necrosis” and is more common in corticosteroid treated patients.

Other joints:

Rheumatoid arthritis affects all the synovial joints. Temporo mandibular involvement produces pain on chewing. Acromio clavicular, sterno clavicular and cricoartenoid joints may also be involved.

Foot Deformities In Rheumatoid Arthritis

- Callosity under PIP joint
- plantar callosity
- Atrophy of plantar metatarsal fat pad
- Prominent metastarsel head
- excessive plantar tilt of metarsals
- claw toes
- hammer toes
- rheumatoid nodules
- calcaneal erosions
- achilles tendinitis
- Flattening of longitudinal arch.
- bunion
- hallux valgus
- overriding of second and third toes
- splaying of forefoot due to divergent metarsals.

Extra – articular manifestations:

Systemic • Fever • Weight loss • Fatigue • Susceptibility of infection	Vasculitis • Digital arteritis • Ulcers • Pyoderma gangreosum • Mononeuritis multiplex • Visceral arteritis
Musculoskeletel • Muscle Wasting • Tenosynovitis • Bursitis • Osteoporosis	Cardiac • Pericarditis • Myocarditis • Endocarditis • Conduction defects • Coronary vasculitis • Granaulomatous aortitis
Haematological: • Anaemia • Thrombocytosis • Eosinophilia	
Lymphatic • Spleenomegaly • Felty's syndrome	Pulmonary • Nodules • pleural effusion • Fibrosing alveolitis • Bronchiolitis • Caplan's syndrome

Nodules • Sinuses • Fistula Occular • Episcleritis • Scleritis • Scleromalacia • Kerato conjunctivitis sicca.	Neurological: • Cervical Cord compression • Compression neuropathies • Peripheral neuropathy • Mononeuritis multiplex • Amyloidosis

DIAGNOSIS

The typical picture of bilateral symmetric inflammatory polyarthritis involving small and large joints in both upper and lower extremities with sparing of the axial skeleton except the cervical spine suggest the diagnosis.

Consititutional features like morning stiffness, demonstration of subcutaneous nodules, presence of the rheumatoid factor and radiographic findings of juxta articular bones substantiate the diagnosis.

Criteria for the diagnosis of Rheumatoid Arthritis:

a. Morning stiffness I hr,
b. Arthritis of 3 or more joint areas.
c. Arthritis of hand Joints.
d. Symmetric Arthritis.
e. Rheumatoid Nodules.
f. Rheumatoid factor.
g. Radiological changes.

Diagnosis of Rheumatoid Arthritis is made with 4 or more criteria. Criteria'a to d' must be present for atleast 6 weeks, and Criteria 'b to e' must be observed by a physician.

INVESTIGATIONS:

A.Haematological:

1. ESR – Increased in active stage
2. Serum proteins – Hyperglobulinaemia with elevation of Gamma and Alpha 2 globulins hypoalbuminaemia during acute phase and C-reactive protein (CRP)

B. Immunological:

i. Rheumatoid Factor (RF):

Latex screening positive

Latex test positive

Sheep cell Agglutination Test (Roose Waaler) (SCAT)

Differential Agglutination Test (DAT)

Human Erythrocyte Agglutination Test (HEAT)

ii Anti Nuclear Antibodies.

Synovial fluid analysis:

Confirms the presence of inflammatory Arthritis. Fluid may show positive Roose-Waaler test in joint fluid, before it can be detected in blood. Also it may show neutrophils or monocytes inclusion bodies.

II. Synovial biopsy:

Villus formation with thickening of synovial layer and infiltration with abnormal cells.

III. Radiographic Evaluation:

- Soft tissue swelling
- Juxta articular osteoporosis
- Erosion of joint margins
- joint spaces are decreased
- deformities
- atlantoaxial subluxation
- subchondralerosions and cyst formation
- fibrous and bony ankylosis develop in the late stages.

IV. Arthroscopy:

In acute Rheumatoid Arthritis synovium is oedematous, diffusely erythematous and friable. In more chronic conditions it becomes thickened.

V. Renal Biopsy:

Indicated in cases of reduced tubular or glomerular function.

VI. Pulmonary Biopsy:

Used to distinguish Rheumatoid nodules from carcinoma or to establish diagnosis of fibrosing alveolitis.

VII. Ultra sound

VIII. Scintigraphy

IX. CT Scanning - shows cartilage and sub –chondral bone damage long before conventional x rays

X. MRI

XI. Urine analysis

XII. Biochemical analysis

XIII. Anti CCP antibodies

(cyclic citrullinated peptide antibodies)

XIV. Genetic tests:

$HLADRB_1$ Typing to detect the presence of 'shared epitope'.

XV. Antinuclear antibody assay (ANA)

XVI. Bone density test to check for bone loss.

DIFFERENTIAL DIAGNOSIS OF RHEUMATOID ARTHRITIS

Sero negative spondyloarthropathy included following rheumatoid like conditions were the serum is negative for rheumatoid factor. They are.

1. Ankylosing spondylitis:

Ankylosing spondylitis is a chronic, progressive and crippling disease affecting the spine. The exact etiology is unclear. Ankylosing spondylitis has been found to be more prevalent in certain races and hence shows a genetic predisposition. It is related to certain tissue types of the human leukocytic antigen (HLA) system. The majority of ankylosing spondylitis patient is found to belong to HLA-B27 groups.

The disease occurs in the 3^{rd} and 4^{th} decades of life and is more common in males. The patients present with complaints of diffuse pain in the back and vague pain in other joints.

2.Reiter's disease:

Reiter's disease characterized by triad of polyarthritis, urethritis, conjunctivitis. The joint condition is an acute polyarthritis resembling rheumatoid arthritis. It does not cause destructive changes in the joint structures. The urethritis is non-gonococcal but the exact organism is not known.

3.Psoriatic arthritis

Psoriatic arthritis is a polyarthritis seen in about 10% of patients with psoriasis.

a. The most common type is the one involving the distal interphalangeal joints of the hands and feet with psoriatic nail joints. Metacarpophalangeal joints are never involved in psoriatic arthritis.

b. Arthritis multilens is a severe form where there is marked destruction of joints.
c. Symmetrical polyarthritic type
d. Oligo arthritic type.
e. Spondyloarthritic type.

4.Enteropathic arthritis

Chronic inflammatory bowel diseases like regional enteritis (Crohn's) disease) and ulcerative colitis are associated with arthritic lesion in abour 10% of the cases. There is peripheral (or) involvement of the spine. The Joint condition shown remissions and exacerbations along with activity of the underlying bowel disease.

5.Sjogren's syndrome:

Sjogren's syndrome is an immunologic disorder characterized by progressive destruction of the exocrine glands leading to mucosal and conjuctival dryness (Sicca syndrome) accompanied by a variety of autoimmune phenomena. Glandular, when the clinical manifestations are within the exocrine system and extra glandular, when other tissues are involved as well.

The disease predominately affects woman in the third of fourth decades of life.

Clinical manifestations:

i. Keratoconjuctivitis and Xerostomia
ii. Renal involvement produces mild interstitial nephritis that may result in renal tubular acidosis.
iii. Sensory polyneuropathy and mononeuritis multiplex.
iv. Pulmonary involvement generally takes the form of an interstitial pneumonitis which is usually a little clinical significance.

COMPLICATIONS

Septic Arthritis:

It may complicate Rheumatoid Arthritis, particularly in patients with long-standing nodular sero-positive disease. In debilitated patients, fever and leucocytosis may be absent and the signs of infection limited to malaise and slight exacerbation of inflammation in one or more joints.

Clinical course and prognosis:

The course of Rheumatoid Arthritis is variable and difficult to predict in an individual patient. Most patients experience persistent but fluctuating disease activity accompanied by a variable degree of joint deformity.

Approximately 15% of patients of R.A. have a short-lived inflammatory process that remits without major deformities.

Complications:

Septic Arthritis:

It may complicate Rheumatoid Arthritis, particularly in patients with long-standing nodular sero-positive disease. In debilitated patients, fever and leucocytosis may be absent and the signs of infection limited to malaise and slight exacerbation of inflammation in one or more joints.

Staphylococcus aureus is commonly implicated secondary to invasion from an ulcerated nodule or infected skin lesion.

Amyloidosis:

It is a complication of prolonged active disease and is formed in 25% to 35% of patients at autopsy, making R.A a leading cause of secondary amyloidosis.

Other than this

1. **Fixed deformities.**

   The perils often the common place ones resulting from ignorance and neglects. Early assessment and planning should prevent postural deformities that will result in joint contractures.

2. **Muscle weakness**

   Even mild degree of myopathy or neuropathy when combined with prolonged inactivity may lead to profound muscle wasting and weakness. This should be prevented by physiotheraphy and pain control if possible. If not the surgeon must be forewarned of the difficulty of postoperative rehabilitation.

3. **Joint rupture**

   Occasionally the joint lining ruptures and synovial contents spill into the soft tissue. Treatment is directed at the underlying synovitis i.e splintage and injection of the joint synovectomy as a second resort.

4. **Spinal cord compression**

   It is a rare complication of cervical spine instability. By the onset of weakness Upper Motor Neuron (UMN) signs in the lower limb is suspicious. If they occur, immobilization of the neck is essential and spinal fusion should be carried out as soon as possible.

5. **Systemic vasculitis**

   This is a rare but potentially serious complication. High doses of corticosteroids and Intra Venous (I.V) plasma volume expanders may be called for.

Juvenile Rheumatoid Arthritis:

Juvenile rheumatoid arthritis (J.R.A) is one of the more common connective tissue diseases of children and is a major cause of functional disability in this age group. By definition, it begins before the age of 16 and most patients are diagnosed early childhood. There is 2:1 female predominance except in the subgroup that has a systemic onset, in which the sexes are equally affected. J.R.A differs from R.A. in adults. In that

1. Oligo arthritis is more common.
2. Systemic onset is more frequent.
3. Larger joints are affected more than smaller joints.
4. Rheumatoid nodules and rheumatoid factor are usually absent and
5. Antinuclear antibody seropositivity is common.

Genetic susceptibility, abnormal immuno regulation, cytokine production and viral infection may all play a role in the pathogenesis. The morphology of the joint, pathology, including marked synovitis is similar to the alteration in adult R.A. The development of symptoms, such as fatigue, joint stiffness and limited range of motion is generally slow and gradual. Commonly targeted joint in all forms of the disease are the knees, wrists, elbows and ankles. They become warm and swollen and are often involved symmetrically, Pericaridtis, myocaridtis, pulmonary fibrosis, glomerulonephritis, uveitis and growth retardation are potential extra-articular manifestations. A systemic onset may begin rather abruptly, associated with high spiking fevers, migratory and transient skin rash, hepatomegaly, spleenomegaly and serositis. Satisfactory recovery occurs in 70 to 90% of patient and in only 10% severe joint deformities present.

MANAGEMENT

Unproven remedies are frequently being sought after and are frequently reported in the lay literature. Calcium and vitamin D have been proven to be beneficial in the treatment of osteoporosis (low bone density). Osteoporosis is frequently seen in patients with rheumatoid arthritis and is a side effect of steroid treatment.

Surgery:

The role of surgery is mainly reconstructive or rehabilitative. The following procedures are used, in the management of Rheumatoid Arthritis.

1. Synovectomy
2. Capsulotomy
3. Osteotomy
4. Arthrodesis
5. Arthroplasty-Excision and Replacement.

Adjunctive treatments:

Rehabilitation:

Restoration of a disabled individual to perform function to a normal or near normal manner is called Rehabilitation.

Rheumatoid Arthritis is a crippling disease. Hence the Rheumatoid Arthritis patients require rehabilitative measures that include physical therapy, and special splints and appliances to help them and manage their own self care activities like dressing and eating.

Physical Therapy (Physiotherapy)

Physiotherapy is the application of physical agents and principles to pathological conditions for the purpose of producing therapeutic effects.

Physiotherapy includes:

1. Active exercise
2. Passive joint movements
3. Local heat
4. Massage
5. Electrical stimulation of muscles
6. Ultra sound therapy
7. Light therapy, ultraviolet rays and infrared rays.

Exercise therapy:

Once inflammation is satisfactorily controlled, appropriate and regular exercises is essential to strengthen muscles weakened by disease. Rheumatoid arthritis often makes joints stiff and restricts their motion if they are not used regularly. Exercises are designed to meet the needs of each patient and should be monitored by professionals specializing in physical medicine.

Exercise guidelines:

1. The patient is advised to engage in low – impact exercises like stationary cycling, rowing and water aerobics and,
2. Start with low intensity exercise.
3. During periods of inflammation or pain (for rheumatoid arthritis), reduce exercise intensity and duration.
4. Patient need longer than normal (longer than ten minutes) warm ups and cool downs.
5. Modify patients exercise intensity or duration depending on how the patient feel that day.
6. It is important that patient should move his joints through a full range of motion at least once a day, otherwise, joints will become stiff.

7. If the patient experience pain two hours after exercise, he need to adjust the intensity or the duration of the exercise.
8. The patients should do exercise to the point in he can (unless he have hypertension) because these type of exercises put the least amount of stress on the joints.
9. Patient should use isometric exercises when he can (unless he have hypertension) because these types of exercises put the least amount of stress on his joints.
10. Advise the patient to consult the doctor if he experience severe pain after exercise.

Active exercise is given to:

Mobilize joints, strengthening muscles, Improve co-ordination or balance.

Passive joint movements:

The chief use of passive joint movement is to preserve full mobility when the patient is unable to move the joint actively.

Types of Exercises:

Range – of – motion Exercise, Strengthening Exercise, Limbering up Exercise:

1. Range – of – Motion Exercises / Stretching Exercises:

Stretching exercise involve moving a joint as it will comfortably go through its full range of motion or stretch. This exercise help to maintain normal joint movement or restore movement that has been lost.

Clinical Assessment of Joint Motion:

The most widely used and recommended instrument is the universal Goniometer, sometimes called an Arthrometer. Basically, it is protractor, to the center of which two long slender arms or levers are

attached. Usually only one of the arms is movable, but may variations in design are possible.

2. Strengthening Exercise:

Strengthening exercises help to maintain or increase the strength and power of the muscles.

3. Limbering up Exercises:

Help to reduce morning stiffness or stiffness after staying in one position too long by doing the Range – of – motion exercises each day only a few times of loosen up.

Details of Range – of – motion Exercises:

Upper Extremities – Shoulder:

1. Arms at side with elbow straight, bring arms forward upward by year.
2. Arms at side with elbow straight, take arms sideward upward.
3. Arms at side bend elbows to right angle and take hands apart.

Elbow:

1. Bend elbow, touching fingers to top of shoulder.
2. Straighten elbow.

Fore arm:

1. Elbows bent, turn palm of the hand and then back of the hand towards face.

Wrist:

1. Keeping forearm steady, move the wrist up and down as in waving.
2. Again hold forearm steady, mover the wrist up and down as in hand shaking.
3. Make circle with hands.

Hand and fingers:

1. Make tight first.
2. Open fingers as wide as possible.
3. With the hand open spread fingers away from each other and then together.
4. Touch tip of the thumb to the tip of each finger.
5. Bend the thumb in toward palm of the hand.

Lower Extremities:

Knee:

Sit with your feet off the floor. Lift the leg and then allow it to return of the bent position slowly.

Ankle:

1. Pull foot up and in, and then push back down.
2. Make circle with foot.
3. Pull foot in toward other foot.
4. Pull foot to outside.

Toes:

1. Pull up on toes then curl toes under.

Exercises for the Neek:

1. In the sitting position, twist your head as far as possible in each direction.
2. Sit or stand with your hands on the hips. First circle the head clockwise, then counter clockwise.
3. In the sitting position, try to touch each shoulder with your head.
4. In the sitting position look behind as far as possible and then look at your toes.

Exercise benefits for individuals with Arthritis:

1. Helps to preserve muscle strength and normal mobility of joints.
2. Relieves pain and stiffness.
3. Prevents further deformities.
4. Improves over-all physical fitness.
5. Improves coordination.

Heat and Cold Treatment:

Head and cold treatment effective means of relaxing muscles and relieving pain in arthritis joints. A hot bath, hat pads, paraffin wax and cold compresses are some methods frequently used.

SELF MANAGEMENT TECHNIQUES FOR RHEUMATOID ARTHRITIS

Self management is the most important aspect of the treatment of Rhematoid arthritis

The Ten Self Help Techniques

1. **Positive mental attitude**

   The patient is told to focus on thins other than pain and their own body. They are encouraged to think positively.

2. **Regular medication:**

   The patient is told the value of regular and correct medication.

3. **Regular exercises :**

   The patient should follow a regular and appropriate exercise programme, most suited for themselves.

4. **Use of joints:**

   The patient is told the value of correct posture and the methods of using the joints.

5. **Energy conservation:**

   Patients are instructed to listen to the body 'inner signals' for rest. Slowing down and avoiding too many activities reduce the stress and strain on the joints.

6. **Assistive devices:**

   Devices like splints, braces and walking sticks can help stabilize the joints, provide strength and reduce pain and inflammation.

7. **Adequate sleep:**

   A good adequate sleep provides rest to the ailing joints and reduces the pain and swelling.

8. **Massage:**

   A Good moderate massage brings warmth and relieves pain due to arthritis.

9. **Relaxation techniques:**

   Relaxation techniques like yoga, meditation, etc, help to relax the muscles, mind and controls respiration heart rate and blood pressure.

10. **Modification in the daily activities**

- ♣ Using western toilets
- ♣ Bath aids and railings
- ♣ long handle broom stick and mob to clean the floors.
- ♣ Use of walking sticks while walking, climbing high chairs
- ♣ Avoid squatting on the ground for food.
- ♣ To avoid squeezing the clothes.
- ♣ To avoid walking on hard and uneven and rough surfaces.
- ♣ To sleep on hard surface.

MATERIALS AND METHODS

The dissertation work on Vali azhal keel vayu was carried out, during, the year of 2006 -2008 at the post graduate department of sirappu Maruthuvam, Govt. Siddha Medical College, Palayamkottai.

Selection of Patients:

For the clinical study 30 patients suffering from Vali azhal keel vayu, were admitted in the inpatient ward of Government Siddha Medical College, palayamkottai of both sexes and varying age groups. Out of these 20 ideal cases were selected for the study. 20 patients were treated as out patients also.

In this study certain criteria was followed based on clinical symptoms, nutritional status, seasonal variations, economic status, family history and other significant diseases. The confirmation of clinical diagnosis was made on both Siddha and modern aspects.

Investigations:

The symptoms of Vali azhal keel vayu more or less correlates with Rheumatoid arthritis in modern medicine. In Siddha aspect the diagnosis was made under the following criteria.

1. Mukkutra nilai
2. Ennvagai thervu
3. Udal kattukal
4. Kalam
5. Nilam
6. Neer kuri
7. Nei kuri

In modern aspects the routine laboratory investigations were made.

Haematological investigations:

1. Total W.B.C. count.
2. Differential W.B.C. count.
3. Erythrocyte sedimentation rate.
4. Haemoglobin percentage.
5. Blood Sugar.
6. Blood Urea.
7. Serum Cholesterol.

Urine Analysis:

1. Albumin
2. Sugar.
3. Deposits

Motion Analysis:

1. Ova
2. Cyst

Specific investigations:

1.Rheumatoid factor.

2.Radiographic evaluation.

Management:

The drugs were selected after a detailed study of various siddha literatures. They are

1. பறங்கிப்பட்டை இராசாயனம் (Parangipattai Rasayanam)
2. கீல்வாதத்திற்கு ஐந்தெண்ணெய் (Keelvathathuku Iynthennai)

The pharmacological study and the bio chemical study of both the drugs were conducted at the Department of pharmacology, Department of BioChemistry, Govt. Siddha Medical College, Palayamkottai respectively.

RESULTS AND OBSERVATION

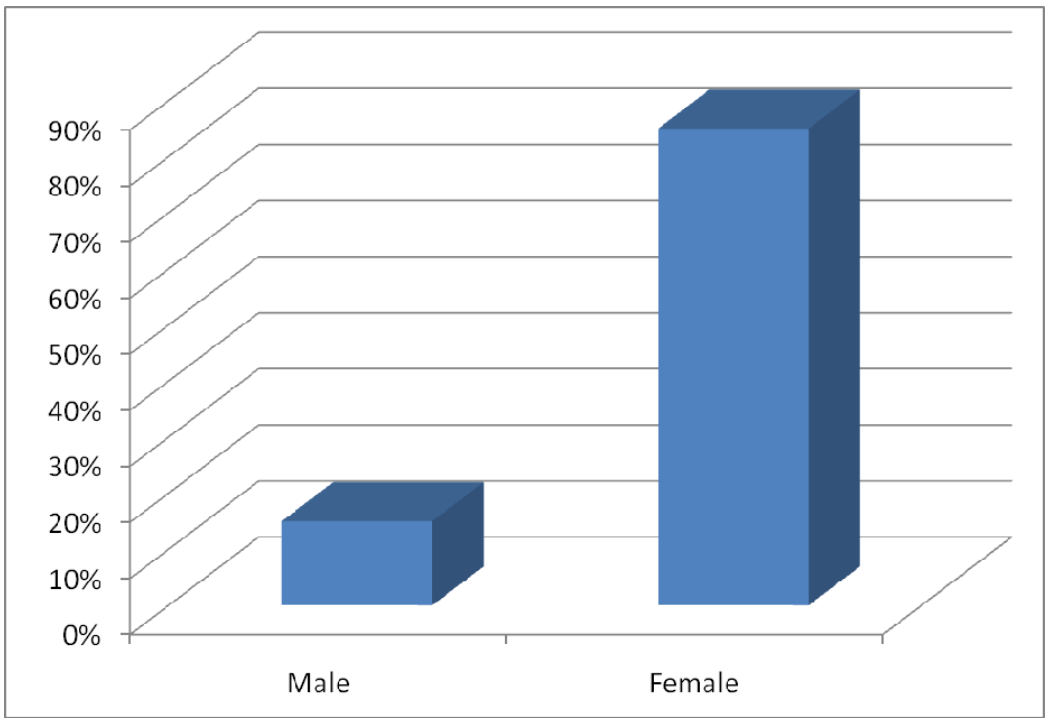
Results were observed with respect to the following criteria.

1. Sex
2. Age
3. Occupation
4. Food habits
5. Economic status
6. Mukkutra kaalam
7. Paruva Kaalam
8. Thinai
9. Mukkutra theory
10. Udal Kattugal
11. Enn vagai thervugal
12. Neikuri
13. Duration of illness
14. Onset of disease
15. Clinical features
16. Results

1.Sex distribution:

S.No.	Sex	No.of Cases (out of 20)	%
1.	Male	3	15%
2.	Female	17	85%
Total		20	100%

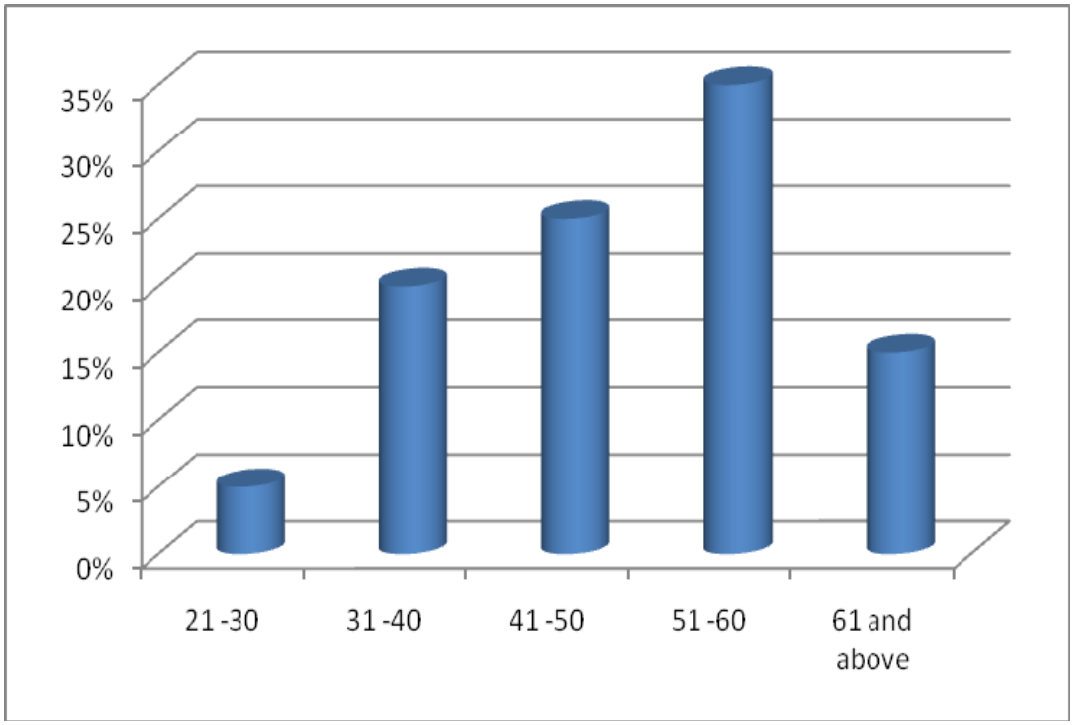
Out of the twenty patients, 3 were males (15%) and 17 (85%) were females.



2.Age distribution:

S.No	Age (inyears)	No.of Cases	%
1.	21 -30	1	5%
2.	31 -40	4	20%
3.	41 -50	5	25%
4.	51 -60	7	35%
5.	61 and above	3	15%

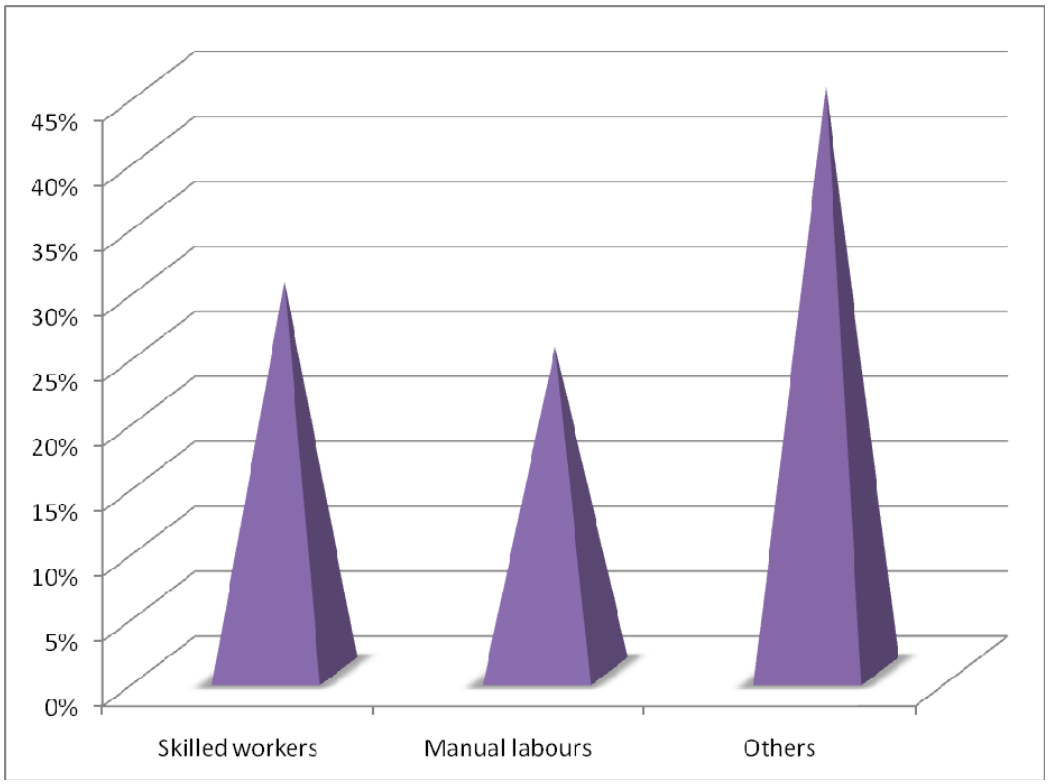
The percentage, was highest in the age group of 51 – 60 (35%) and 25% in 41 and above and the least percentage was between 21 -30 (5%) among the studied cases.



3.Occupation:

S.No	Occupation	No.of Cases	%
1.	Skilled workers	6	30%
2.	Manual labours	5	25%
3.	Others	9	45%

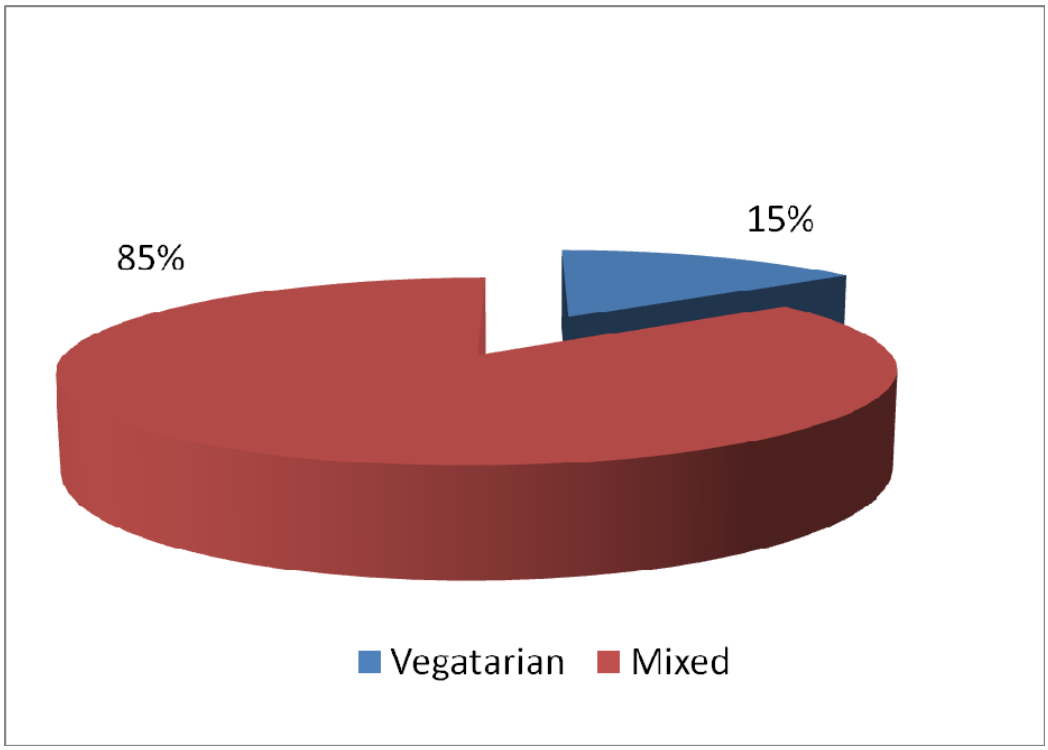
The percentage of occupation was 30% among skilled workers, 25 % were manual labours and 45% were others.



4. Food Habits:

S.No	Food Habits	No.of Cases	%
1.	Vegatarian	3	15%
2.	Mixed	17	85%

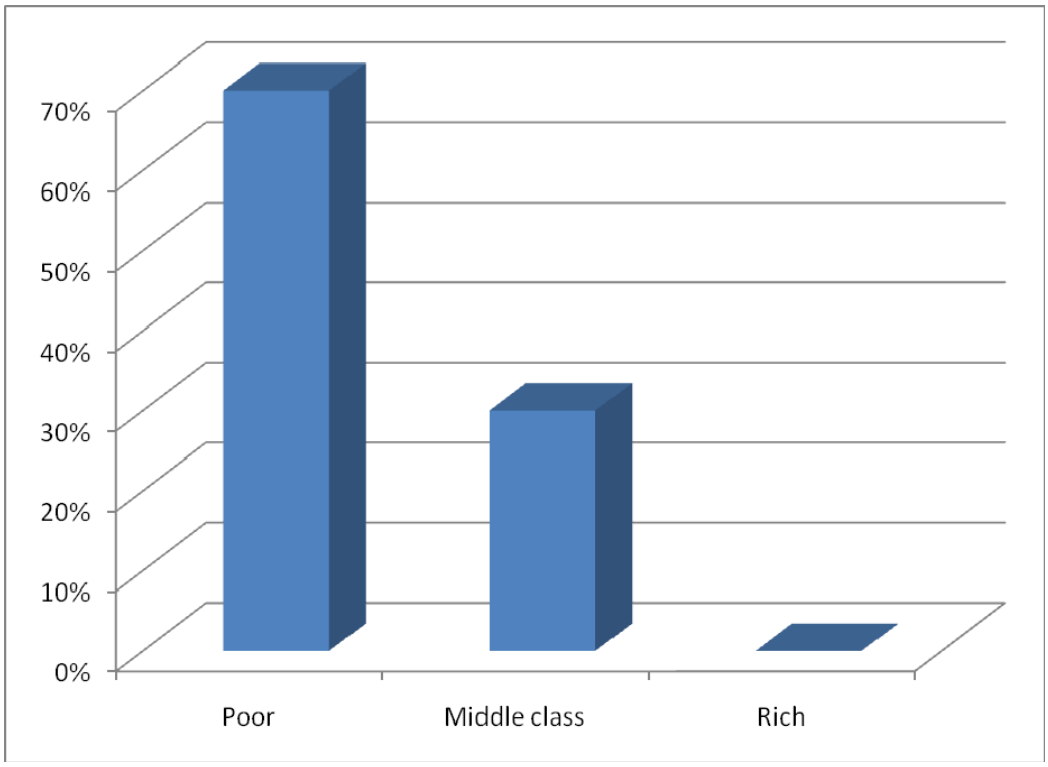
According to food habits, 85 % of the cases had mixed diet and 15% were vegetarians.



5. Socio – economic Status:

S.No	Socio economic status	No.of Cases	%
1.	Poor	14	70%
2.	Middle class	6	30%
3.	Rich	-	-

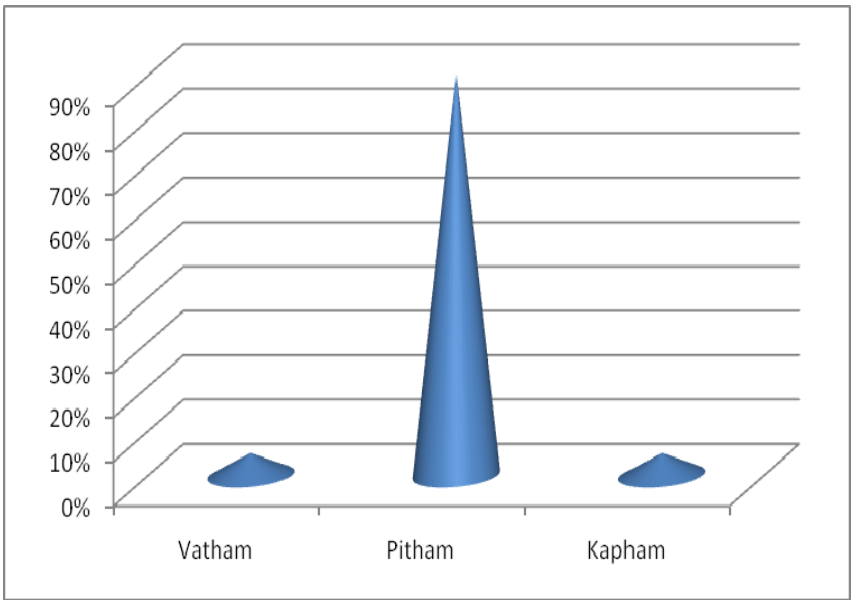
According to the study, 70% of the cases belonged to poor class economic status.



6. Mukkutra kaalam:

S.No.	Kalam	No.of cases	Percentage	Total percentage
1.	Vatham			
	0-11	-	-	5%
	11-22	-	-	
	22-33	1	5%	
2.	Pitham			
	33-44	4	20%	90%
	44-55	6	30%	
	55-66	8	40%	
3.	Kapham			
	66-77	1	5%	5%
	77-88	-	-	
	88-100	-	-	

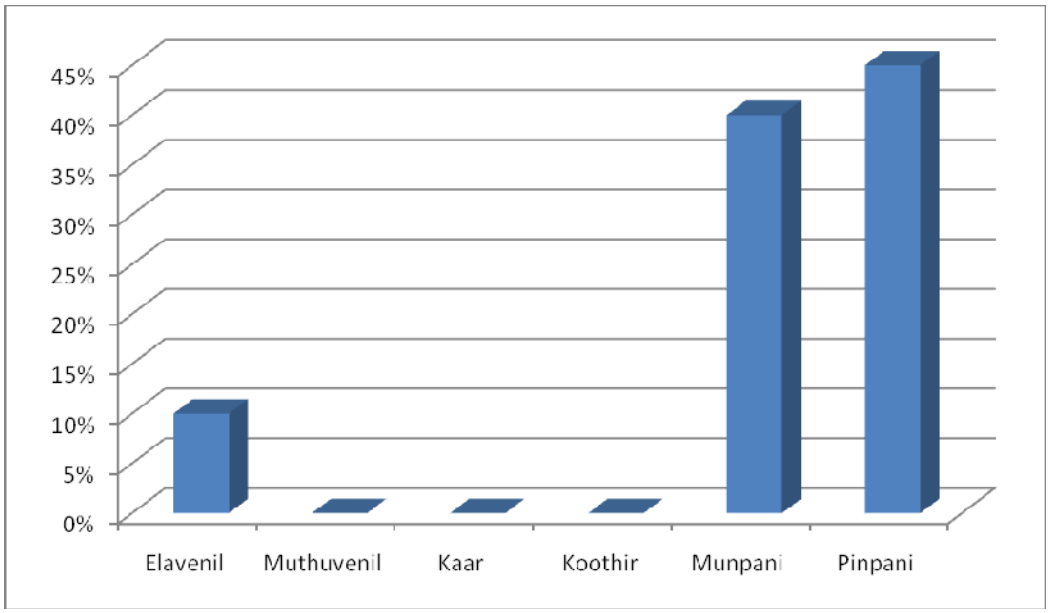
Among the twenty cases, 90% of the cases were under pitha kaalam and 5% of the cases were under vatha kalam and 5% were under kaba kalam.



7.Paruva kaalam:

S.No.	Paruva kaalam	No.of Cases	%
1.	Elavenil (chithirai – Vaikasi)	2	10%
2.	Muthuvenil (Aani – Aadi)	0	-
3.	Kaar (Aavani – Purattasi)	0	-
4.	Koothir (Ippasi – Kaarthikai)	1	5%
5.	Munpani (Markazhi – Thai)	8	40%
6.	Pinpani (Maasi – Panguni)	9	45%

Under paruva kalam, the highest incidence 45% was noted in pinpani kalam and 40% in Munpani kalam.



8.Thinai

S.No.	Thinai	No.of Cases	%
1.	Kurinji	-	-
2.	Mullai	-	-
3.	Marutham	20	100
4.	Neithal	-	-
5.	Paalai	-	-
Total		20	100

All the twenty cased belonged to Marutha nilam

9.Mukkutra theory:

A. Derangement in the types of vatham:-

S.No.	Types of Vatham	No.of Cases	%
1.	Pranan	-	-
2.	Abanan	10	50
3.	Viyanan	20	100
4.	Uthanan	4	20
5.	Samanan	15	75
6.	Naagan	-	-
7.	Koorman	-	-
8.	Kirukaran	-	-
9.	Devathathan	10	50
10.	Dananjeyan	-	-

According to the derangement in the types of vatham, 50% of the cases abanan was affected, viyanan was also affected in 100% of the cases, samanan was also affected in 75% of the cases and devathathan were affected in 50% of the cases and uthanan in 20% of cases.

B.Derangement in types of pitham:

S.No.	Types of Pitham	No.of Cases	%
1.	Analam	10	50
2.	Ranjakam	12	60
3.	Saathakam	15	75
4.	Prasakam	5	25
5.	Aalosakam	-	-

Among the cases studied, analam pitham was affected in 50% of the cases and saathaka pitham was affected in 75% of the cases and prasakam was affected in 25% of the cases and Ranjakam was affected in 60% of cases.

C. Derangement in types of kabam:

S.No.	Types of Kabam	No.of Cases	%
1.	Avalambakam	-	-
2.	Kilethakam	10	50
3.	Pothakam	-	-
4.	Tharpakam	-	-
5.	Santhikam	20	100

Among the 20 cases, kilethakam were affected in 50% of cases, santhikam were affected in 100% of the cases.

10. Udalkattugal

S.No.	Udalkattugal	No.of Cases	%
1.	Saaram	20	100
2.	Senneer	12	60
3.	Oon	3	15
4.	Kozhuppu	-	-
5.	Enbu	20	100
6.	Moolai	-	-
7.	Sukkilam/ Suronitham	-	-

Among the 20 cases, saaram and enbu were found affected in 100% of the cases and senneer was affected in 60% of the cases and in 15% of the cases oon was affected.

11. Ennvagai thervugal:

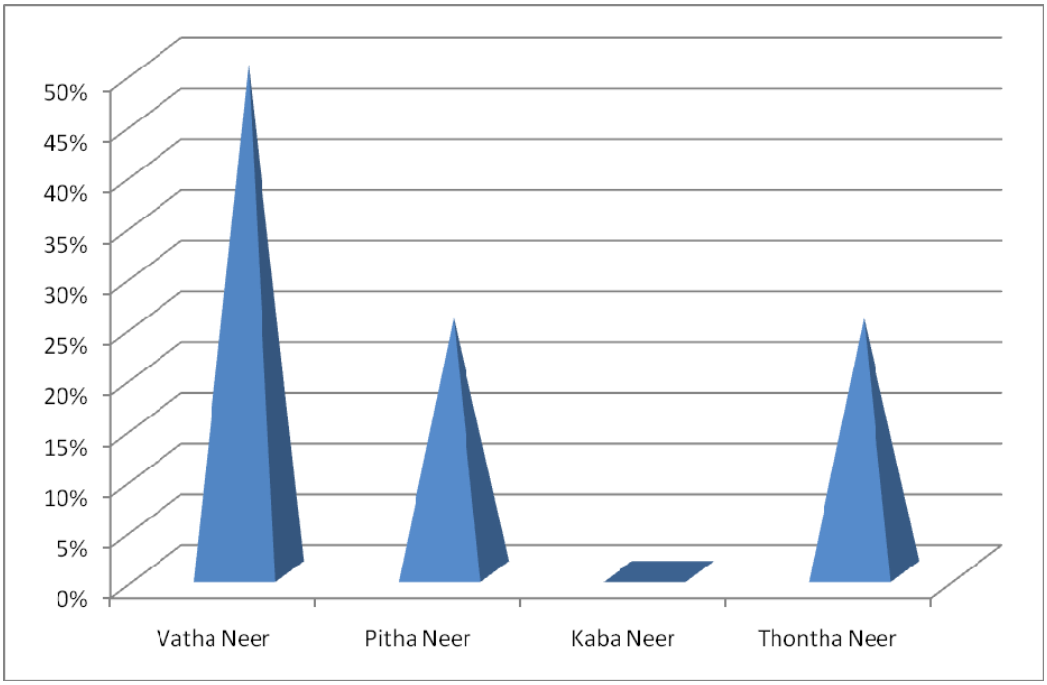
S.No.	Enn Vagai Thervugal	No.of Cases	%
1.	Naa	5	25
2.	Niram	10	50
3.	Mozhi	-	-
4.	Vizhi	10	50
5.	Sparism	20	100
6.	Naadi	20	100
7.	Malam	10	50
8.	Moothiram	5	25

Among the cases studied, sparism, naadi and were affected in 100% of the cases. Niram, Vizhi, Malam were affected in 50% of the cases and naa, moothiram were affected in 25% of the cases.

12. Neikuri:

S.No.	Character of Urine	Neikuri Reference	No.of Cases	%
1.	Spreads like snake	Vatha Neer	10	50
2.	Spreads like ring	Pitha Neer	5	25
3.	Spreads like pearl	Kaba Neer	-	-
4.	Spreads like snake in ring	Thontha Neer	5	25

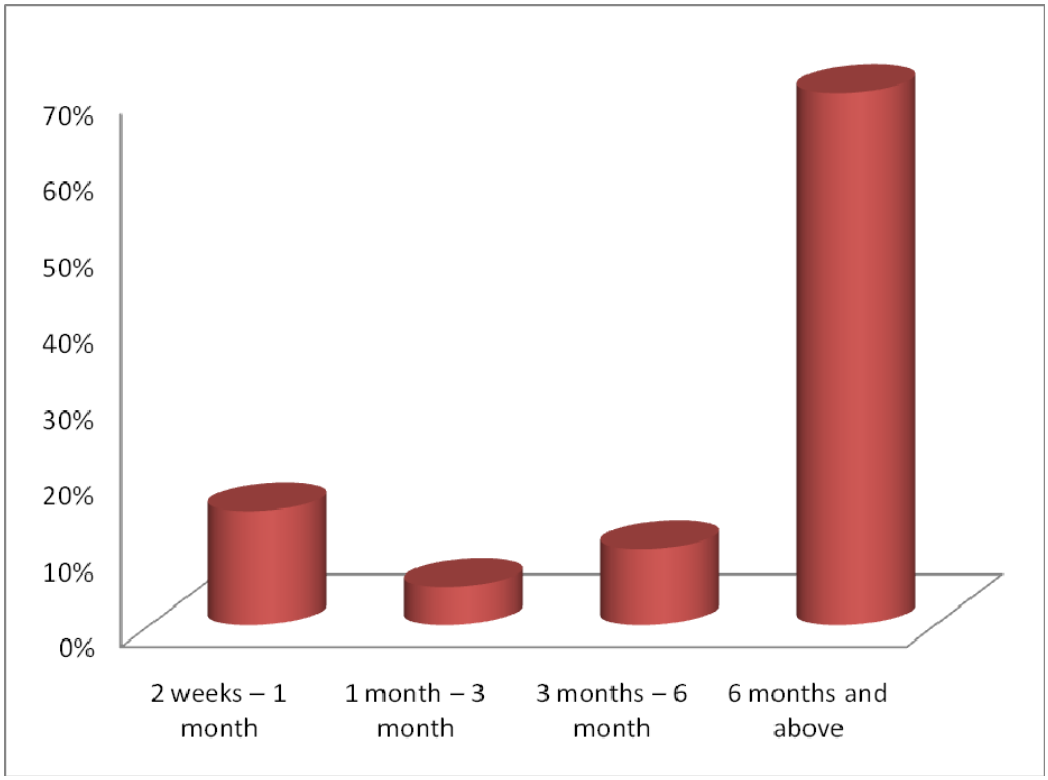
Neikuri in 50% of the cases was Vatha neer and 25% of the cases were both Pitha neer and Thontha neer respectively.



13. Duration of the illness:

S.No.	Duration of illness	No.of Cases	%
1	2 weeks – 1 month	3	15
2.	1 month – 3 months	1	5
3.	3 months – 6 months	2	10
4.	6 months and above	14	70

Out of cases, 70% of the cases were affected for the more than 6 months and 5 % were less than 3 months.



14. Onset of the disease:

S.No	Mode of onset	No. of cases	%
1.	Sudden	3	15%
2.	Gradual	17	85%

According to the study, 85% of the cases had gradual onset of disease and 15% had sudden onset.

15. The clinical features: (Symptoms)

S.No.	Symptoms	No.of Cases	%
1.	Joint pain	20	100
2.	Swelling	20	100
3.	Morning stiffness	20	100
4.	Restricted movements	15	75
5.	Difficulty in chewing	2	10
6.	Difficulty in walking	8	40
7.	Neck pain	3	15
8.	Sleeplessness	10	50
9.	Fever	6	30
10.	Loss of appetite	10	50
11.	Loss of weight	3	15
12.	Constipation	10	50
13.	Giddiness	3	15

Among the 20 patients, 100 % of the cases had joint pain, swelling and morning stiffness. 75 % of the cases had restricted movements.

Clinical features (Signs)

S.No.	Signs	No.of Cases	%
1.	Subcutaneous nodules	1	5
2.	Muscle wasting	3	15
3.	Opthalmic manifestation	-	-
4.	Hepatomegaly	-	-
5.	Spleenomegaly	-	-
6.	Anaemia	10	50
7.	Tenderness	20	100

Out of the 20 Cases, 100% of the cases had tenderness and 50% of the cases were anaemic and 15% of the cases had muscle wasting and 5 % of the cases had subcutaneous nodules.

Grading of pain, joint swelling and restricted movements:

S.No.	Symptoms	No.of Cases			%
		Mild	Moderate	Severe	
1.	Pain	3(15)	10 (50)	7 (35)	100
2.	Joint swelling	2(10)	13 (65)	5 (25)	100
3.	Restricted movements	2(10)	10 (50)	3 (15)	75
4.	Muscle wasting	3 (15)	-	-	15

All the 20 Cases(100%) had pain and joint swelling and 75% with restricted movements and 15% of the cases had muscle wasting.

Involvement of locomotor system

Table showing involvement of extremities

S.No.	Mode of onset		No.of Cases	%
	Early	Late		
1.	Upper extremities	Lower extremities	15	75
2.	Lower extremities	Upper extremities	3	15
3.	Both extremities		2	10

Out of the 20 Cases 75 % of the cases had involvement of the upper extremities first and then the lower extremities. 15% of the cases had involvement of the lower extremities first and then the upper extremities. In 10% of the cases both extremities were involved.

The joint involvement:

S.No.	Joints involved	No.of Cases	%
1.	Proximal interphalangeal joints of hand.	20	100
2.	Metacarpophalangeal joints	20	100
3.	Wrist joint	15	75
4.	Elbow	4	20
5.	Shoulder	2	10
6.	Temporomandibular jt	2	10
7.	Sternoclavicular jt.	3	15
8.	Cervical spine	3	15
9.	Hip joint	-	-
10.	Knee joint	10	50
11.	Ankle Joint	12	60
12.	Metatarsophalangeal Jt	2	10

Out of the cases, 100 % of the cases had proximal interphalangeal joint and metacarpophalangeal joint involvement. 75 % had wrist joint involvement. 60% had ankle joint involvement, knee joint was involved in 50% of the cases. 15% had cervical and claviclar Joint involvement. 10% had shoulder, temporomandibular and metatarsophalangeal joint involvements.

Table showing deformities of the joints

S.No.	Deformities	No.of Cases	%
1.	Interphalangeal joints	2	10
2.	Metacapophalangeal joints	2	10
3.	Wrist joint	2	10
4.	Elbow joint	-	-
5.	Shoulder joint	-	-
6.	Hip joint	-	-
7.	Knee joint	-	-
8.	Ankle joint	-	-
9.	Metatarsopahalangeal joint	-	-
10.	Spine	-	-

Out of the 20 cases studied 10% of the cases had deformities in the wrist joint, meta carpophalangeal joint and interphalangeal joint.

The Grading of Rheumatoid arthritis:

Grade I - No restriction of ability to perform normal activities

Grade II - Moderate restriction but with ability to perform most of the daily activities.

Grade III - Marked restriction with an inability to perform daily activities.

Grade IV - Incapacitation with confinement to bed.

Table for grading of R.A.

S.No.	Grade	No.of Cases	%
1.	I	3	15
2.	II	10	50
3.	III	7	35
4.	IV	-	-

Among the 20 Cases, 50% of the cases were under grade II and 35% of the cases belonged to grade III and 15% of the cases belonged to grade I.

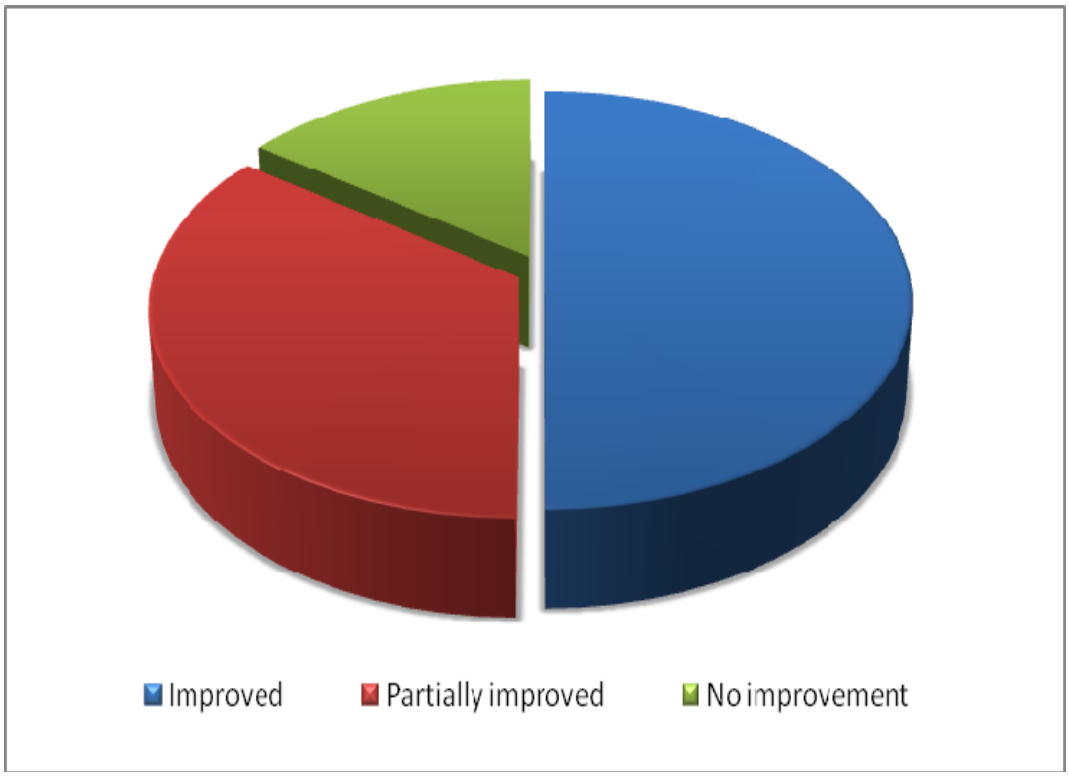
16.Results:

S.No.	Results	No.of Cases	Percentage
1.	Improved	10	50
2.	Partially improved	7	35
3.	No improvement	3	15

Improved : Complete subsidence of pain and Swelling

Parially Improved : Relief of pain, reduction in swelling and increased range of movements

No Improvement :- Persistance of pain, Swelling and other signs and symptoms.



DISSCUSSION

According to Siddha system of medicine diseases are classified in to 4448 types . Among them Yudi vaidhaya chinthamani has classified vatha diseases into 80 types. Among the 80 types, 10 varieties are concerned with joint diseases. One among them is Santhu vatham. In Sabapathy scripts this disease has been compared with the disease keel vayu, explained in Siddha Maruthuvam text.

Keel vayu is classified into 10 types. Vali azhal keel vayu which is the study topic of the author is one among the 10 types. Thirty cases of Valiazhal keelvayu were admitted in the Post graduate sirappu maruthuvam inpatient ward, GSMC palayamkottai. Out of these twenty cases were selected for this study.

For diagnostic purposes the parameter used in siddha aspect were (porial arithal, pulanal arithal, Vinaathal, Kanmendriyam, Gnanendriyam, uyir thathukkal, Udal thathukal, Ennvagai thervugal, Neerkuri, Neikuri etc.) The modern parameters used were criteria for the diagnosis of Rheumatoid arthritis, laboratory investigations, radiological evaluation etc.

The sex distribution:

Out of the twenty patients 3 (15%) cases were males and 17 were females (85%)

The age distribution:

Among the 20 cases, 5% belonged to the age group of 21 -30 years. 20% of the cases were between 31 -40 years. 25% of the cases were between 41 -50 years. 35 % of the cases were between 51 -60 years 15% of the cases were above 61 years.

From the above data, the incidence of the disease seems to be more in the age group of 51 -60 years, and the next is between 41 -50 years.

The kaalam:

The patients in Vaatha kalam were 5%. 90% of the patients were of pitha kalam and 5% of the cases were of kaba kalam. According to Siddha concept maximum number of cases were noted to be in pitha kalam which is between 33 to 66 years, which co – ordinates with modern concept of the incidence in 4 th – 5th decade.

The paruva kalam:

Among the 20 cases 45% of the cases were seemed to develop the disease during pinpani kalam (Maasi – panguni) 40% of the cases Munpani kalam (Markazhi – thai) and 10% of the cases developed during Elavenil (chithirai – vaikasi) 5% of cases developed during koothir (Ippasi – Kaarthikai)

The thinai:

All the 20 cases were belonged to Marutha nilam. Even though as per siddha literatures, Marutha nilam is said to be free from diseases, here the patients developed diseases due to alteration in their food habits and routine activities.

The socio – economic status:

70% of the cases were under poor class, where as 30% of the cases belonged to middle economic groups.

The aetiological factors:

10% of the cases had positive family history.Literary evidences shows that Vali azhal keel vayu develops during winter and has genetic predisposition.

The onset of disease:

The mode of onset was acute in 15% and gradual onset in 85% of the cases. Thus the incidence of the disease with gradual onset is more, as per literatures.

The clinical manifestations:

The patients with Valiazhal keelvayu (R.A.) present with articular manifestations with or without extra articular manifestations were noticed.

100 % of the cases had joint pain, swelling and morning stiffness.75% of the cases had restricted movements and 40% of the cases had difficulty in walking. 15% of the cases had pain in the neck. The joint in all the cases experienced early morning stiffness for bout 2-3 hours. Decreased movements of the joints noticed in all the cases which was assessed by asking the patient to move the joint in particular direction

Involvement of upper and lower extremities:

The incidence of initial involvement of joints of the upper limb was noticed in 75 % of the cases. 15 % of the cases, showed involvement of the lower limbs while 10% of the cases showed involvement of upper and lower limbs simultaneously.

Involvement of individual joints:

The proximal interphalangeal joint and meta carpophalangeal joints were involved in 100% of the cases. 75 % of the cases wrist joint. 60% of the cases ankle joint. 50% of the cases the knee joint, 15% of the cases cervical and sternoclavicular joint and 10% of the cases shoulder, temperomandibular and metatarsophalangel joints were involved.

Deformities of joints:

10% of the cases had deformities in the wirst joint, metacarpophalangeal joint and interphalangeal joint.

Elicitation of extra articular manifestations:

Besides the articular symptoms certain signs which include the examination of the enlargement of liver and spleen were carried out. Examination of respiratory system, cardio vascular system, were also carried out. Opthalmic manifestation were also noted. Either hepatomegaly or spleenomegaly was not present in any of the cases. Likewise respiratory and cardio vascular manifestation and ophthalmic manifestation were not present. 5% of case had rheumatoid nodule.

Uyir thathukkal:

Uyir thathukkal include 3 vital humours namely

Vatham

Pitham

Kabam

The derangement in any of the above three causes diseases. They were noticed in the 20 cases and are discussed below.

Vatham:

Viyanan was affected in 100% of the cases. Abanan was affected in 50% of the cases Samanan was affected in 75 % of the cases.

When abanan is affected it produces constipation, the disturbance in viyanan produce pain and restricted movements of the joints and if samanan is affected there is loss of appetite.

Udhanan was affectd in 20% of the cases and devathathan was affected in 50% of the cases

Pitham:

Anala pitham was affected in 50% of the cases. Saathaga pitham affected in 75% of the cases and ranjakam was affected in 60% of the cases. Prasakam was affected in 25% of the cases.

Affected anala pitham produce loss of appetite. Ranjakam, when affected produces decrease in haemoglobin and affected saathaga pitham produce restriction of movements

Kabam:

Santhigam was affected in 100% of the cases. Kilethagam was affected in 50% of the cases.

Affected kilethagam produce loss of appetite and affected santhigam produce joint pain, swelling and restriction of movements.

Udalkattugal:

Saaram and Enbu were affected in 100% of the cases which shows general debility and restricted movements, joint pain and swelling.

Senneer was affected in 60% of the cases which shows reduced haemoglobin percentage. Oon was affected in 15% of the cases which shows, slight muscular wasting.

Ennvagai thervgal:

Naadi was observed in all the 20 cases. All the cases had thontha naadi.

Sparism was affected in all the cases which showed joint tenderness, warmthness over the inflammed joints.

Malam was affected in 50% of cases and they had constipation.

Naa was affected in 25% of the cases and the tongue was coated and was pale in colour due to anaemia.

Niram and vizhi were affected in 50% of the cases and showed anaemic manifestations.

Neikuri:

50% of the cases showed vaatha neer and 25% of the cases had pitha neer and 25% of the cases had thontha neer.

Neer kuri was found straw in colour which indicate for Vali azhal keel vayu.

Grading of Vali azhal keel vayu patients:

Grading of Rheumatoid arthritis was useful to assess the severity of the disease.

Grade 1:

No restriction of ability to perform normal activities.

Grade II:

Moderate restriction but with an ability to perform most of the daily activities.

Grade III:

Marked restriction with an inability to perform most of the daily activities.

Grade IV:

In capactitation with confinement to bed.

15% of the cases belonged to Grade I

50% of the cases belonged to Grade II

35% of the cases belonged to Grade III

Investigations:

Besides siddha based investigations since the disease has been compared with Rheumatoid arthritis, investigations meant for rheumatoid arthritis were also done. Routine examination of blood, urine and stools were done during the admission and the discharge.

Examination of urine and stools showed no abnormalities.

Examination of blood showed 50% of the cases showed decreased in haemoglobin percentage and 95% of the cases had increased ESR with maximum of 100 mm in I hour.

Rheumatoid arthritis factor was positive in thirteen cases.

Blood sugar, Blood urea and Serum cholesterol were done in the Biochemistry Department Govt. Siddha Medical College, Palayamkottai. The values were found normal in all the cases.

The patients were also subjected to radiological evaluation which revealed R.A.

The management:

Siddha system of medicine follows certain criterias in the management of the diseases.

“Viraesanathal vatham thazhum”

This shows that before treating vatha diseases, laxatives or purgatives can be administered to correct altered vatham.

Valiazhal keelvayu comes under vatha diseases, all the 20 cases were given 5 gms of Nilavagai choornam with hot water at bed time on the first day of admission. The next day the following medicines were started.

The internal drug given was **“பறங்கிப்பட்டை இராசயனம்”** (Parangipattai Rasayanam) 1 to 2 gms, external drug given was **“கீல்வாதத்திற்கு ஐந்தெண்ணெய்”** (Keelvathathirku Iynthennai) two times daily. Clinically the drugs were free from side effects. The patients were asked to apply the oil over the swellings and in the areas of pain.

Hot water fomentation was adviced to all the patients after the application of the oil. Since this may increased the blood supply to that area.

In the initial stage all the patients were advised to take bed rest. After the severity of symptoms had reduced, they were advised to do some exercises.

The patients were observed carefully when they were performing the exercises. Care was taken for patients to perform exercise slowly without any strain.

Assessment of the effects of management:

All the 20 cases were treated with **"பறங்கிப்பட்டை இராசயனம்"** (Parangipattai Rasayanam) internally and**"கீல்வாதத்திற்கு ஐந்தெண்ணெய்"** (Keelvathathirku Iynthennai) externally. Thokkanam was also advised. The results of treatment was assessed on the basis of reduction in symptoms such as pain, joint swelling, and a sense of well being. The patients were treated for about 10 to 40 days. At the end of the treatment the results were discussed as follows: Improved, Partially improved and no improvement.

50% of the cases improved well.

35% of the cases were partially improved and

15% of the cases showed no improvement.

At the end of the treatment there was a rise in Hb level and decreased in ESR level in all the cases. After discharge the patients were advised to attend the post graduate department of Sirappu maruthuvam O.P. for further follow up.

During the course of treatment diet restrictions was strictly advised. Patients were advised to take preventive measures like avoiding exposure to chill weather. They were advised to take bath in warm water.

SUMMARY

Valiazhal keelvayu is one among a common problem in clinical practice. It has received an international attention in search of finding out of a new drug for the ailment of the sufferers.

Valiazhal keelvayu is a chronic multi system connective tissue disorder of unknown aetiology. The characteristic feature of Rheumatoid arthritis is persistant inflammatory synovitis usually involve the peripheral joints in a symmetrical fashion. The potential of the synovial inflammation to cause cartilage destruction and bone erosion and subsequent joint deformity is the hall mark of the disease.

Joint pain, Swelling, tenderness which may be aggravated on motion. Morning stiffness of 2 to 3 hours duration, easy fatigability, anaemia, occasional fever are the common manifestation of the disease.

Extra articular manifestations are Rheumatoid nodules, lymph adenopathy, Spleenomegaly, Rheumatoid vasculitis, ophthalmic manifestations include scleritis, kerato conjunctivitis and pleuritis, fibrous alveolitis.

The study on Valiazhal keelvayu (R.A) was with trial drugs.

1. **"பறங்கிப்பட்டை இராசயனம்"** (Parangipattai Rasayanam) 1-2 gms B.D. Internal.
2. **"கீல்வாதத்திற்கு ஐந்தெண்ணெய்"** (Keelvathathirku Iynthennai) External.

Twenty cases of both sexes, and of various age groups were chosen and the study was carried out.

90% of the patients belonged to pitha kaalam. 45% of the cases were seemed to develop the disease during pinpani kaalam (maasi – panguni). All the cases came from Marutha nilam. Seventy percent of

cases belonged to poor class families. Considering aetiology ten percent of the cases had a positive family history.

85% of the cases had a gradual on set of disease. All the cases had joint pain, swelling, morning stiffness. 75% of the cases had restricted movements. 75% of the cases had initial involvement of joints of the upper limb.

In all the cases proximal interphalangeal joints and meta carpophalangeal joints were involved. Ten percent of the cases had deformities in their interphanlangeal, metacarpophalangeal joints and wrist joint.

Regarding uyir thathukkal, viyanan and was were affected in 100% of the cases abanan was affected in 50% of cases samanan was affected in 75% of cases anala pitham was affected in 50% of the cases. In 60% of the cases ranjakam was affected and in 75% of the cases sathaka pitham was affected. And santhigam were affected in all the cases, kilethagam was affected in 50% of cases.

Saaram and enbu were affected in all the cases. Senneer was affected in 60% of the cases. Regarding Enn vagai thervugal, sparism. Naadi were affected in all the cases. Neerkuri was found straw in colour. Neikuri indicated Vatha neer in 50% of the cases.

Malam was affected in 50% of cases.

50% of the cases had moderate restriction but with an ability to perform normal activities.

R.A. factor was positive in thirteen cases. 50% of the cases showed a decrease in haemoglobin percentage. E.S.R. was raised in all the cases. The trial medicines were given to 20 Cases with Valiazhal keel vayu.

The internal drug was **“பறங்கிப்பட்டை இராசயனம்”** (Parangipattai Rasayanam) in the dose of 1-2 gms twice a day after food.

The external drug was **”கீல்வாதத்திற்கு ஐந்தெண்ணெய்”** (Keelvathathirku Iynthennai) which was applied externally to the affected joints.

Hot water fomentation was given after the application of external drug.

All the patients were advised to follow the balanced dietary habit.

All the patients were also advised to follow the preventive measures like avoiding exposure to cold weather and advised to take bath in warm water.

The observation made during this study, showed that the trial medicines were clinically effective.

The potency of the drugs were studied by pharmacological and biochemical analysis. It shows that the drugs has acute anti inflammatory, anti pyretic and analgesic actions.

CONCLUSION

In this study, results were found to be good in 50% of the cases.

No adverse effects were noticed during the treatment period.

Further follow up of these patients showed good recovery and fine improvement in the general well beings as they could carry out their day to day activities.

The preparation of both medicines are simple and expiry is six months for Rasayanam and one year for Iynthennai. So they can be stored and used.

The trial medicine has acute anti inflammatory, analgesic and antipyretic actions.

Early diagnosis and prompt management prevents the disabilities and deformities resulting from Rheumatoid arthritis.

So it is concluded that the treatment with **“பறங்கிப்பட்டை இராசயனம்”** (Parangipattai Rasayanam) **”கீல்வாதத்திற்கு ஐந்தெண்ணெய்”** (Keelvathathirku Iynthennai) is good in the view of efficacy and safety.

PREPARATION OF TRIAL DRUGS

பறங்கிப்பட்டை ரசாயணம்

சேரும் சரக்குகளும் அளவுகளும்:

சுத்தித்த பறங்கிப்பட்டை	-	300 கிராம்
சுத்தித்த கந்தகம்	-	140 கிராம்
சுத்தித்த குக்கில்	-	140 கிராம்
சுக்கு	-	25 கிராம்
மிளகு	-	25 கிராம்
திப்பிலி	-	25 கிராம்
கடுக்காய் தோல்	-	25 கிராம்
தான்றிக்காய் தோல்	-	25 கிராம்
நெல்லி முள்ளி	-	25 கிராம்
இலவங்கப்பட்டை	-	25 கிராம்
சிறுநாகப்பூ	-	25 கிராம்
அதிவிடயம்	-	25 கிராம்
நற்சீரகம்	-	25 கிராம்
கருஞ் சீரகம்	-	25 கிராம்
ஓமம்	-	25 கிராம்
கடுகு ரோகிணி	-	25 கிராம்
குரோசாணி ஓமம்	-	25 கிராம்
கொடிவேலி வேர்ப்படை	-	25 கிராம்
சுத்தித்த சிவதை வேர்	-	25 கிராம்
சுத்தித்த அரிதாரம்	-	25 கிராம்
நவாச்சாரம்	-	25 கிராம்
பொரித்த வெங்காரம்	-	25 கிராம்
குந்தரிக்கம்	-	25 கிராம்
சீனி சர்க்கரை	-	900 கிராம்

செய்முறை:

சுத்தி செய்த குக்கிலை கொஞ்சம் பசுவின் நெய் தெளித்து வறுத்திடித்து தூள் செய்து கொள்ளவும். மற்ற சரக்குகளையும் சுத்தித்து தனி தனியே சூரணித்து ஒன்றாக கலந்து நன்றாயரைத்து சீனிச்சர்க்கரையும் சேர்த்து பீங்கானில் பத்திரபடுத்தி வைத்து கொள்ளவும்.

அளவு : 1-2 கிராம் மூன்று வேளை

அனுபானம் : பால், தேன்

தீரும் நோய்கள் : கீல்வாதம் தீரும்

ஆதார நூல் : அகத்தியர் வைத்திய பிள்ளை தமிழ்

- பக்க எண் 108

PROPERTIES OF TRIAL DRUGS

1. **பறங்கிப்பட்டை (Smilax china)**

சுவை : இனிப்பு

தன்மை : தட்பம்

பிரிவு : இனிப்பு

குணம் :

“ தாகம் பலவாதந் தாதுநட்டம் புண்பிளவை
மேகங் கடிகிரந்தி வீழ்மூலந் - தேகமுடன்
குட்டை பகந்தமேற் கொள்வமனம் போம்பறங்கிப்
பட்டையினை யுச்சரித்துப் பார் ’’

-தேரையர் குணவாகடம்

தீரும் நோய்கள் :

பல விதமான வாத நோய்கள், உடல் தாதுக்கள் குறைதல், மேகநோய், புண் பிளவை.

2. கந்தகம் (Sulplur)

சுவை : கைப்பு, துவர்ப்பு

குணம் :

“ நெல்லிக்காய் கந்திக்கு நீள்பதிணென் குட்டமந்தம்
வல்லை கவிசைகுன்ம வாயுகண்ணோய் - பொல்லா
விடக்கடிவன் மேகநோய் வீறுசுரம் பேதி
திடக்கிரக ணீகபம்போந் தேர் ’’

- பதார்த்த குண சிந்தாமணி

தீரும் நோய்கள் :

பதிணெண்குட்டம், மந்தம், மேகநோய், வாதத்தினால் வந்த சுரம், குன்மவாயு, பேதி, கிரகணி.

3. குக்கில் (Shorea robusta)

சுவை : கைப்பு

தன்மை : வெப்பம்

பிரிவு : கார்ப்பு

குணம் :

" காததர நாசிநோய் கட்டிகடி மேகப் புண்
வாத வித்தி ரஞ்சூலை வன்குநகம் - ஓதிவையித்
திக்கி லிருக்கத் திடமுள்ள வோவுபயக்
குக்கி லிருக்கநமக் குள் ''

- பதார்த்த குண சிந்தாமணி

தீரும் நோய்கள் :

காது, உதடு, மூக்கு ஆகிய இடங்களிலுண்டாகும் நோய்கள், மேகப் புண், நிணக்கழிச்சல், கீல் பிடிப்பு.

4. சுக்கு (Zingiber officinale)

சுவை : கார்ப்பு

தன்மை : வெப்பம்

பிரிவு : கார்ப்பு

குணம் :

" வாதப் பிணிவயி நூதற் செவிவாய்
வலி தலை வலி குலை வலியிரு விழிநீர்''

- பதார்த்த குண சிந்தாமணி

தீரும் நோய்கள் :

வாதப் பிணிகள், வயிற்றுப்பிசம், தலைவலி, குலை வலி, அன்மை பார்வை கெடுதி.

5. மிளகு (Piper nigrum)

சுவை : கைப்பு கார்ப்பு,

தன்மை : வெப்பம்,

பிரிவு : கார்ப்பு

குணம் :

" அளவையுறாக்காரம் அடைந்திருக்கும் வாத
விளைவையெல் லாமனுக்கும் மெய்யே – மிளகின்வாய்
கண்டவர்க்கும் இன்பமாம் காரிகையே சீழ்மூலவ்
கொண்டவர்க்கு நன்மருந்தாய் கூறு''

- பதார்த்த குண சிந்தாமணி

தீரும் நோய்கள் : வாத நோய்கள், சீழ்மூலம்

6. திப்பிலி (Piper longum)

சுவை : இனிப்பு

தன்மை : தட்பம்

பிரிவு : இனிப்பு

குணம் :

" வரும லப்பெருக் கோடு மகோதரம்
வாதம் ஆதிமுத் தோடஞ் சுரங்குளிர்
பெருமாலைப் புரி மேகப் பிடகமும்
பேருந் திப்பிலிப் பேரங்குரைக்கவே''

- பதார்த்த குண சிந்தாமணி

தீரும் நோய்கள் :

வாத நோய்கள், சுரம், குளிர், மகோதரம், ஈளை, இருமல், இரைப்பு

7. கடுக்காய் தோல் (Terminalia arjuna)

சுவை : முக்கிய சுவை துவர்ப்பு அத்துடன் இனிப்பு, புளிப்பு. கார்ப்பு, கைப்பு

பிரிவு : இனிப்பு

குணம் :

" பழமலத்தைப் போர்க்கும் பகரிலுடலுக்
கழகு தரும் புத்தி யளிக்கும் - பழகி
மருவ் கடுத்த வாதபித்த வன்கபத்தை தீர்க்குங்
கருங்கடுக்கா யென்றுளத்திற் காண்''

- பதார்த்த குண சிந்தாமணி

தீரும் நோய்கள் :

பழைய மலத்தை போக்கும், உடலுக்கு அழகையும் புத்திக்கு தெளிவையும் உண்டாக்கும். வாத பித்த கபத்தை நீக்கும்.

8. தான்றிக்காய் தோல் (Terminalia bellirica)

சுவை : துவர்ப்பு

தன்மை : வெப்பம்

பிரிவு : இனிப்பு

குணம் :

" சிலந்திவிடம் காமியப்புண் சீழான மேகங்
கலந்தவரும் வாதபித்தவ் காலோ – மலர்ந்தடலில்
ஊன்றிக்காய் வெப்ப முதிரபித் துங்கரக்குந்
தான்றிக்காய் கையிலெடுத் தால்''

- பதார்த்த குண சிந்தாமணி

தீரும் நோய்கள் :

வாத நோய்கள், மேக நோய்கள், சிலந்தி விடம், ஆண்குறி புண்

9. **நெல்லிமுள்ளி (Phyllanthus emblica)**

சுவை : புளிப்பு, துவர்ப்பு, இனிப்பு

தன்மை : தட்பம்

பிரிவு : இனிப்பு

குணம் :

"நெல்லிக்காய்க்குப் பித்தம் நீங்கு மதன்புளிப்பால்
செல்லுமே வாதமிதற் சேர்துவரால் - சொல்லுமையம்
ஓடுமிதைச் சித்தத்தில் உள்ள அனலுடனே
கூடுபிற மேகமும் போங் கூறு "

- பதார்த்த குண சிந்தாமணி

தீரும் நோய்கள் :

வாத, பித்த கப நோய்களினால் உண்டாகும் கேடு, வாந்தி, சுவையின்மை, மேகம்.

10. **அதிவிடயம் (Aconitum heterophyllum)**

சுவை : கைப்பு

தன்மை : வெப்பம்

பிரிவு : கார்ப்பு.

தீரும் நோய்கள் :

முறை காய்ச்சல், கழிச்சல், வாதம், மூலம், மூலக்கடுப்பு போக்கும்.

11. **சீரகம் (Cuminum cyminum)**

சுவை : கார்ப்பு

தன்மை : தட்பம்

பிரிவு : இனிப்பு

தீரும் நோய்கள் :

வயிற்றுவலி, வாய் நோய், குருதி கழிச்சல், கல்லடைப்பு வளி நோய்கள் விலகும்.

12. **கொடிவேலி (Plumbago indica)**

சுவை : கார்ப்பு

தன்மை : வெப்பம்

பிரிவு : கார்ப்பு

"சூலையும் வாய்வுஞ் சுகசன்னி மாந்தசுரஞ்
சீலசிர நோய்வாதந் தீருவ்காண் - கோலமயிந்
பேடனைய மாதே பிறங்குசெஞ்சித் ரம்மூலம்
சேடு செம்பைத் துய்யபற்பஞ் செய்".

- பதார்த்த குண சிந்தாமணி

தீரும் நோய்கள் :

சூலை, மாந்தம். வாதம், சுகசன்னி.

13. **கடுகுரோகினி (Picrorhiza scrophulariiflora)**

சுவை : கைப்பு கார்ப்பு

தன்மை : வெப்பம்

பிரிவு : கார்ப்பு

தீரும் நோய்கள் :

மாந்தம், சுரம், ஐயப்பெருக்கு, வளி நோய்கள்.

14. **சிவதை (Operculina turpethum)**

சுவை : கைப்பு

தன்மை : வெப்பம்

பிரிவு : கார்ப்பு

"உள்ள மலமும் உதவார்த்தமும் வயிற்றைக்
கொள்ளு பித்த வாதமும்போவ் கூறுங்கால்"

- பதார்த்த குண சிந்தாமணி

தீரும் நோய்கள் :

வயிற்றுன்னுண்டாகும் கேடுகள், வாத நோய்களும் தீரும்.

15. அரிதாரம்

"சுரங்குளிர்மா வாதமுடற் சூலைநமை குட்டம்
இரங்குமென நாளும் இசை"

- பதார்த்த குண சிந்தாமணி

தீரும் நோய்கள் :

சுரத்துடன் கூடிய குளிர், வாதநோய், உடலில் குத்தல், நமைச்சல், குட்டம்.

16. நவாச்சாரம்

"சன்மகவிச் சுமுத தோடவ் கனவாத நீங்கும்
நவாச்சார மாதே நவில்"

- பதார்த்த குண சிந்தாமணி

தீரும் நோய்கள் :

முத்தோடம், வாத நோய்கள்.

17. வெங்காரம்

தீரும் நோய்கள் :

தவளை சொறி, எண்வகை குன்மம், பங்குவாதம், சந்நிபாதம்.

18. இலவங்கப்பட்டை (Cinnamomum verum)

சுவை : காரம், இனிப்பு

தன்மை : தட்பம்

பிரிவு : இனிப்பு

தீரும் நோய்கள்

இரைப்பு, இருமல், வீக்கம்,தீரும்

19. **இலவங்கப்பத்திரி (Cinnamomum Tamala)**

சுவை : கார்ப்பு

தன்மை : வெப்பம்

பிரிவு : கார்ப்பு

தீரும் நோய்கள்

சுரம், நீர்வேட்கை, வாந்தி

20. **சிறுநாகப்பூ** (Mesua ferra)

சுவை : சிறுகைப்பு, துவர்ப்பு

தன்மை : தட்பம்

பிரிவு : கார்ப்பு

தீரும் நோய்கள்

வெள்ளை, இருமல், வாதநோய்கள் தீரும்

21. **ஓமம்** (Carum coptium)

சுவை : கார்ப்பு

தன்மை : வெப்பம்

பிரிவு : கார்ப்பு

தீரும் நோய்கள்

குடலிரைச்சல், வாயு,சிரங்கு நீங்கும்.

22. **குரோசாணி ஓமம்** (Hyoscyamus niger)

சுவை : கார்ப்பு, சிறுகைப்பு

தன்மை : வெப்பம்

பிரிவு : கார்ப்பு

தீரும் நோய்கள்

தூக்கமின்மை, சூதகவாயு, வாத நோய்கள் நீங்கும்.

23. **குந்திரிக்கம்** (Boswellia serrata)

சுவை : கைப்பு

தன்மை : வெப்பம்

பிரிவு : கார்ப்பு

தீரும் நோய்கள்

நாட்பட்ட புண்கள், வீக்கம் தீரும்.

24. **கருஞ்சீரகம் (Nigella sativa)**

சுவை : கைப்பு

தன்மை : வெப்பம்

பிரிவு : கார்ப்பு

தீரும் நோய்கள்

தலை நோய், இருமல், வீக்கம் தீரும்

வெளி மருந்து

கீல்வாதத்திற்கு ஐந்தெண்ணெய்

நல்லெண்ணெய்	-	500	மில்லி லிட்டர்
தேங்காய் எண்ணெய்	-	500	மில்லி லிட்டர்
விளக்கெண்ணெய்	-	500	மில்லி லிட்டர்
வேப்பெண்ணெய்	-	500	மில்லி லிட்டர்
பசு நெய்	-	500	மில்லி லிட்டர்
எருக்கு இலை	-	5	

செய்முறை:

மேற்கண்ட எண்ணெய்களை ஒன்று சேர்த்து இரும்பு கடாயிலிட்டு அடுப்பேற்றி காய்ச்சவும்.

எண்ணெய் காய்ந்தவுடன் எருக்கு இலைகளை ஒவ்வொன்றாக எண்ணெயிலிட்டு பொரித்து எடுத்து கொள்ளவும்.

தீரும் நோய்கள்:

சர்வ வாதங்களும் போகும்.

ஆதார நூல் :

சித்த மருந்து செய்முறைகள்

-பக்க எண் 83

1.**வேப்பெண்ணெய் (Azadirachta indica)**

சுவை : கைப்பு

தன்மை : வெப்பம்

பிரிவு : கார்ப்பு

குணம் :

“வாதம் போம்.....

.....

வேப்ப நெய் யென்றொருக்கால் விள்ளு”.

- பதார்த்த குண சிந்தாமணி

தீரும் நோய்கள் :

வேம்பின் நெய்யை பூச, பெரும் வளி நோய் வகைகள் போகும்.

2.எள்நெய் (Sesamum indicum)

சுவை : இனிப்பு

தன்மை : வெப்பம்

பிரிவு : இனிப்பு

குணம் :

“செப்பரிய வாதபித்தத் தீமையகலும்”

- பதார்த்த குண சிந்தாமணி

தீரும் நோய்கள் :

உடல்வன்மை தரும். வாத பித்த நோய்கள் தீரும்.

3.ஆமணக்கெண்ணெய் (Ricinus communis)

சுவை : கைப்பு

தன்மை : வெப்பம்

பிரிவு : கார்ப்பு

குணம் :

“ஆமணக்கெண்ணெய்……...

பூமணச் சந்துதோறும் பொருந்திய வாதம் போகும்”

- பதார்த்த குண சிந்தாமணி

தீரும் நோய்கள் :

குழந்தைகளை தாய் போல் வளர்க்கும் வாதநோய்கள் போகும்.

4.எருக்கு (Calotropis gigantea)

சுவை : கைப்பு, காரம், மதுரம்.

தன்மை : வெப்பம்

பிரிவு : கார்ப்பு

“ எலி விடங் குட்டமைய மேறு கிருமி

வலி சூலை வாயு விட மந்தம்”

- பதார்த்த குண சிந்தாமணி

தீரும் நோய்கள்

ஐவகை வலி, கீல்வீக்கம், வளி நோய்கள் போகும்.

BIO - CHEMICAL ANALYSIS OF PARANGIPATTAI RASAYANAM

Preparation of the extract:

5gms of Choornam was weighed, accurately and placed in a 250ml clean beaker. Then 50ml distilled water is added and dissolved well. Then it is boiled well for about 10 minutes. It was cooled and filtered in a 100ml volumetric flask and then it is made up to 100ml with distilled water. This fluid is taken for analysis.

Qualitative analysis:

S.No	Experiment	Observation	Inference
1.	TEST FOR CALCIUM 2ml of the above prepared extract is taken in clean test tube. Add 2ml of 4% Ammonium oxalate solution is added to it.	A white Precipitate is formed	Indicates the presence of caluim is present
2.	TEST FOR SULPHATE: 2ml of the extract is added to 5% barium chloride solution.	No white Precipitate is formed	Absence of Sulphate
3.	TEST FOR CHLORIDE The extract is treated with silver nitrate solution	No white Precipitate is formed	Absence Of Chloride
4.	TEST FOR CARBONATE: The substance is treated with concentrated HCL.	No brisk Effervessence is formed	Absence of Carbonate

5.	TEST FOR ZINC: The extract is added with Potassium ferro cyanide solution.	No white precipitate is formed	Absence of Zinc
6.	TEST FOR IRON FERRIC: the extract is treated with glacial acetic acid and potassium Ferro cyanide.	No blue Colour is formed	Absence of ferric Iron.
7.	TEST OF IRON FERROUS: The extract is treated with concentrated Nitric acid and ammomuin thio cynate:	Blood Red colour is formed	Indicates the presence of ferrous Iron
8.	TEST FOR PHOSPHATE The extract is treated with ammonium Molybdate and concentrated nitric acid	No yellow Precipitate is formed	Absence of phosphate
9.	TEST FOR ALBUMIN The extract is treated with Esbach's reagent	No yellow Precipitate is formed	Absence of Albumin
10.	TEST FOR TANNIC ACID The extract is treated with ferric chloride reagent	Blue black Precipitate is formed	Indicates the presence of Tannic acid

11.	TESTFOR UNSATURATION Potassium permanganate solution is added to the extract	It gets decolourised	Indicates the presence of unsaturated compound
12.	TEST FOR THE REDUCING SUGAR: 5ml of Benedict's qualitative solution is taken in a test tube and allowed to boil for 2 mts and added 8-10 drops of the extract and again boil it for 2 mts.	Colour change occurs	Indicates the presence of reducing Sugar
13.	TEST FOR AMINO ACID: One or two drops of the extract is placed on a filter paper and dried it well after drying 1% Ninhydrin is sprayed over the same and dried it well.	No Violet colour is formed	Indicates the Absence of Amino acid

ANALGESIC STUDY OF PARANGIPATTAI RASAYANAM

Aim

To study the effects of analgesic action on Albino rats by Tail - Flick method.

Instruments

Dolonometer (or) Analegesic using heated nicrome wire a source of stimulus.

Procedure

6 albino rats were selected on either sex and divided into 2 groups. Each group having 3 rats were weighting between 100 – 150 gm. The rat is holded on the instrument. So that the tail lies over the nichrome wire on analgesiometer without touching it. To heat the nicrome wire by switching it on and at the same time starting a stop watch. The time takes for the rat to flick the tail was noted. This is the reaction time. This was noted for each rat and the average was calculated. This is kept as control volume.

Paracetamol was administered at a dose of 20 mg / 100 mg of body weight orally to the second group of rats, the reaction time was noted after the administration of ½ hr. and one hour and the average is calculated. The test does was given for the third group of rats.

The result of test drug is compared with the standard as well as control group

S.No.	Group	Dose-100 gm of body weight	Initial Readings in second	After ½ hr in second	After 1 hr in second	After 1 ½ hr in second	Mean Difference
1.	Control	Water – 2ml	2 Secs	2 Secs	2.5 Secs	2.5 Secs	2.5 Secs
2.	Standard	Paracetamol – 20 mg	2 Secs	3 Secs	3.5 Secs	6.5 Secs	6.7 Sec
3.	Test drug	Parangipattai Rasayanam 100mg	2 Secs	2.5 Secs	3 Secs	5 Secs	5 Sec

Inference:

From the above tabulation it is noted that Parangipattai Rasayanam has Significant analgesic action.

ANTI-PYRETIC STUDY ON PARANGIPATTAI RASAYANAM

Aim

To study the anti-pyretic activity of the Parangipattai Rasayanam.

Procedure

Three groups of healthy albino rats were taken, each weigh about 100-200gm and divided into three groups, each group consists of 2 rats. All the rats were made hyperthermic by subcutaneous injection of 12% suspension of Yeast at a dose of 100ml/100mg body weight.

10 hrs later one group of rats were given the test drug Parangi pattai Rasayanam at a dose of 1 ml at a dose of 100 mg/100 gm of body weight. The other group received distilled water at a dose of 1 ml/100 gm of body weight and kept as control. The last group was given Paracetamol at a dose of 100 mg/ 100 gm body weight and kept as standard.

The mean rectal temperature for 3 groups was recorded at 0 hr, ½ hr, 1 hr, 1 ½ hrs after the drug administration. The difference in mean temperature between the 3 groups were noted and compared.

Group	**Dose volume orally/100gm bw**	**Mean temperature**			
		Initial	**½ hr**	**1 hr**	**1 ½ hr**
Control	1 ml	37.0	37.0	37.0	37.0
Paracetamol	20 mg	37.5	37.0	35.0	34.0
Parangi pattai Rasayanam	100mg	37.0	36.0	35.5	35.5

Inference - The test drug Parangi pattai Rasayanam has moderate anti pyretic action.

ACUTE ANTI-INFLAMMATORY ACTIVITY IN RATS BY HIND-PAW METHOD

To demonstrate the acute anti-inflammatory activity of Parangi pattai Rasayanam in albino rats by Hind-paw method.

Procedure:

Nine Albino rats weighing 100-150gm were taken and divided into three groups and each group consisting three rats.

First group was kept as control and received water. Second group received Ibuprofen at a dose of 20mg/100gm-body weight. Third group animals received Parangi pattai Rasayanam suspension at a dose of 200mg/100gm-body weight.

Before administration of drugs, the Hind-paw volume of all the rats were measured. This was done by dipping the Hind-paw upto the tibio-tarsal junction in mercury plethysmograph. Soon after the measurement at the drugs were administered. One hour after the administration of drugs a sub-cutaneous injection of **0.1ml** of 1%/W/V of carrageenin in water was made into planter surface of both the Hind-paw of each rat.

Three hours after carrageenin injection, the Hind-paw volume was measured once again. Difference between the initial and final value were noted and compared.

This method is more suitable method for studying anti-inflammatory activity in acute inflammation.

The effect of Parangipattai Rasayanam in acute anti-inflammatory activities.

Group	Dose volume orally	Initial reading	Final reading	Mean difference	Percentage Inflammation	Percentage Inhibition
Control	Water 2 ml	0.55	1.4	0.85	100	-
Standard	Ibuprofen 20mg/100gm	0.55	0.85	0.3	35.2	64.8
Test drug	200mg/100gm	0.7	0.95	0.25	29.7	70.6

Result:

The drug Parangipattai Rasayanam has Significant acute – anti inflammatory action.

CHRONIC ANTI-INFLAMMATORY STUDY BY COTTON-PELLETS GRANULOMA METHOD

Drug

Parangipattai Rasayanam.

Aim

To study the chronic anti-inflammatory activity of the drug in albino rats by cotton pellets implantation (granuloma) method.

Procedure:

Cotton pellets each weighing long was prepared and sterilized in an autoclave for about one hour under 15 lbs atmosphere pressure. Nine Albino rats weighing between 100-200gm were selected and were divided into 3 groups. Each rat was anaesthetized with ether and cotton pellets were implanted subcutaneously in the groin, two in each side.

From the day of implantation, one group of animals received Parangipattai Rasayanam. at a dose of 200mg/100gm of body weight.

On the eighth day the rats were sacrificed and the pellets were removed weighed. Then they were put in an incubator at 60ºC-80ºC and then weighed. The concordant weight was noted for all groups and compared.

The effect of Parangipattai Rasayanam **in chronic anti inflammatory study.**

Group	**Dose given orally**	**Concordant weight in mgs**	**Percentage inflammation**	**Percentage inhibition**
Control	Water 2ml	250	100	-
Standard	Ibuprofen 20mg/100gm body weight	55	22	78
Test drug	200mg/100gm body weight	110	42	58

Inference :

The drug show Significant chronic – anti inflammatory action.

ACUTE ANTI – INFLAMMATORY STUDY ON KEELVATHATHIKKU IYNTHENNAI

(EXTERNAL USE)

By Hind – Paw method in Albino Rats

Aim

To study the acute antiinflammatory activity of the test drug Keelvathathikku Iynthennai.

Procedure

Six healthy albino rats weighing 100 -150 gm, were taken and divided into two groups, each consisting of 3 rats.

First group was kept as control by giving distilled water 2 ml/ 100 gms of body weight. The second group was kept as test group.

Before application of test drug, the hind – paw volume of all rats were measured. This was done by dipping the hind – paw (upto tibiotrasal junction) into a mercury plethysmography. While dipping the hind paw, by pulling the syringe piston, the level of mercury in the ventre small tube was made to coincide with red marking and reading was noted from the plethysmograph.

One hour later, a sub – cutaneous injection of 0.1 ml of 1% (W/V) carrageenin in water was made into plantar surface of both hind – paw of each rat. To the second test group keelvathathikku Iynthennai. was topically applied for three times over the inflamed surface in a thin layer with 30 minutes gap. To the other groups no drug was applied over the inflamed surface.

One and half hour after injection. The hind – paw volume was measured once again. The difference between the initial and final volume would show the amount of inflammation. Taking the volume in the

control groups as 100% of inflammation, anti – inflammatory effect of the test group is calculated.

Effect of Keelvathathikku Iynthennai:

S.No.	Group	Dose/100gm Of body weight	Mean Difference	Percentage of Inflammation	Percentage of inhibition
1.	Control	Water -2 ml	0.85	100	-
2.	Standard	2 ml	0.3	35.2	64.8
3.	Test drug Keelvathathikku Iynthennai	External	0.22	25.8	74.2

Inference:

It is observed that Keelvathathirku Iynthennai has Significant acute anti inflammatory action.

PROFORMA OF CASE SHEET

GOVERNMENT SIDDHA MEDICAL COLLEGE & HOSPITAL

POST GRADUATE DEPARTMENT

PALAYAMKOTTAI. TRIRUNELVELI – 627 002

Branch – III Sirappu Maruthuvam

Dissertation done by:

I.P. No	:	Occupation	:
Bed No	:	Income	:
Ward	:	Nationality	:
Name	:	Religion	:
Age	:	Date of Admission	:
		Date of Discharge	:
Sex	:	Diagnosis	:
		Result	:
		Medical Officer	:

Permanent Address :

Complaints and duration :

History of present illness :

History of Previous illness :

Personal history including habits:

Family History :

GENERAL CONDITIONS ON EXAMINATION

1. Consciousness :

2. General appearance :

3. Stature :

4. Nourishment :

5.Skin Changes :

6. Face :

7. Pallor :

8. Jaundice :

9. Cyanosis :

10. Clubbing :

11. Lymphadenopathy :

12. Abdominal Distension :

13. Jugular Venous Pulsation :

14. Engorged Veins :

15. Koilonychia :

16. Pedal oedema :

17. Generalised Oedema :

18. Temperature :

19. Pulse :

Rate :
Rhythm :
Volume :
Character :
peripheral pulses :
Pulses paradoxus :

20. Respiratory Rate : /min

21. Heart Rate :

22. Blood pressure :

	Right	Left
Upper limb		

23. Miscellaneous :

சித்த முறை தேர்வு

1. நிலம்

   குறிஞ்சி

   முல்லை

   மருதம்

   நெய்தல்

   பாலை

2. பருவ காலம்

   கார் காலம் (ஆவணி - புரட்டாசி)

   கூதிர் காலம் (ஐப்பசி - கார்த்திகை)

   முன்பனி (மார்கழி - தை

   பின்பனி (மாசி- பங்குனி)

   இளவேனில் (சித்திரை - வைகாசி)

   முதுவேனில் (ஆனி - ஆடி)

3. யாக்கை (உடல்)

   வாதம்

   பித்தம்

   கபம்

   கலப்பு

4. குணம்

   சத்துவம்

   இராசதம்

   தாமசம்

5. பொறி புலன்கள்

   மெய் - ஊறு

   வாய் - சுவை

   கண் - ஒளி

   மூக்கு - நாற்றம்

   செவி - ஒலி

6. கன்மெந்திரியம்

கை - தானம்

கால் - கமனம்

வாய் - வசனம்

வருவாய் - விசர்க்கம்

கருவாய் - ஆனந்தம்

7. உட்காயம்

தோள் பொருத்து

புயம்

முழங்கை பொருத்து

சயம்

கைக்குளசு

கரபம்

அங்குலி

அங்குசம்

8. அதக்காயம்

இடுப்பு பொருத்து

தொடை

முழங்கால் பொருத்து

முன்கால்

குதிங்கால்

கரபம்

அங்குலி

வம்சி

9. மும்மலம்

மலம் -

மூத்திரம் -

வியர்வை -

10. பிற உறுப்புகளின் நிலை

இருதயம் -

புப்புசம் -

இரைப்பை -

கல்லீரல் -

மண்ணீரல் -

சிறுகுடல் -

பெருங்குடல் -

சிறுநீரகம் -

மூளை -

கருப்பை -

11. உயிர் தாதுக்கள்

(அ) வாதம் -

பிராணன் -

அபானன் -

வியானன் -

உதானன் -

சமானன் -

நாகன் -

கூர்மன் -

கிருகரன் -

தேவதத்தன் -

தனஞ்செயன் -

(ஆ) பித்தம்

அனற் பித்தம் -

இரஞ்சக பித்தம் -

சாதக பித்தம் -

ஆலோசக பித்தம் -

பிராசக பித்தம் -

(இ) கபம்

அவலம்பகம் -

கிலேதகம் -

போதகம் -

தற்பகம் -

சந்திகம் -

12. உடல் தாதுக்கள்

சாரம் -

செந்நீர் -

ஊண் -

கொழுப்பு -

என்பு -

மூளை -

சுக்கிலம்/சுரோணிதம் -

13. எண் வகைத்தேர்வுகள்

நாடி -

ஸ்பரிசம் -

நா -

நிறம் -

மொழி -

விழி -

மலம்

நிறம் -

எடை -

இறுகல் -

இளகல் -

சிறுநீர்

1 நீர்க்குறி

நிறம் -

மணம் -

எடை -

நுரை -

எஞ்சல் -

2. நெய்க்குறி

LABORATORY INVESTIGATION

1. BLOOD:

TC : cells/ cu. mm

DC : P % L % E % M %

ESR :

½ hour : mm

I hour : mm

Hb :

Blood Sugar :

Blood Urea :

Serum Cholesterol :

Uric Acid :

VDRL :

2. URINE

Albumin :

Sugar :

Deposits :

3. Motion

Ova : Cyst :

4. IMMUNOLOGICAL :

RA Factor :

5.RADIOGRAPHIC FINDINGS :

6. SEROLOGICAL TEST FOR SYPHILIS :

7. SYNOVIAL FLUID ANALYSIS :

8. ARTHROGRAPHY :

1. LOCOMOTOR SYSTEM

INSPECTION

Overlying Skin:

Colour :

Scars and ulcers :

Periarticular swelling :

Bones:

Deformity :

Unusual posture :

Muscle changes :

Symmetrical distribution :

joint movements :

Gait :

Palpation:

Skin Temperature :

Soft Tissues :

Bony Enlargement :

Crepitus :

Sub-cutaneous nodules :

Rheumatoid vasculitic lesion :

Lymphadenopthy :

pitting Iedema :

Range of Movements :

Examination of individual joints:

Cervical Spine :

Thoracic Spine :

Lumbar Spine :

Sacro-illac Joint :

Shoulder Joint :

Elbow Joint :

Wrist Joint :

Metacarpophalangeal joint :

Interphalangeal Joint :

Hip Joint :

Knee joint :

Metatarso phalangeal Joint:

Examination of individual joints (Affected Joints)

Measurement (in inches) :

Before and After Treatment:

Affected Joints	**RIGHT**		**LEFT**	
	BEFORE	**AFTER**	**BEFORE**	**AFTER**
Knee Joint Ankle Joint Wrist Joint Elbow joint Index finger Middle finger Ring finger Little finger				

EXAMINATION OF OTHER SYSTEMS:

1. Respiratory system :
2. Cardio Vascular system :
3. Gastro Intestinal system :
4. Central Nervous system :

DAILY PROGRESS

DATE	SYMPTOMS	DRUG

GOVERNMENT SIDDHA MEDIDCAL COLLEGE HOSPITAL,

POST GRADUATE DEPARTMENT,

PALAYAMKOTTAI, TIRUNELVELI-627 002

BRANCH – III SIRAPPU MARUTHUVAM.

ADMISSION-DISCHARGE SHEET FOR "VALI AZHALKEEL VAYU"

IP No :

Bed No :

Ward :

Name :

Age :

Sex :

Permanent Address:

Temporary Address:

Occupation :

Income :

Nationality :

Religion :

Date of Admission :

Date of Discharge :

Diagnosis :

Result :

Medical officer :

CLINICAL PICTURES

Sl.No.	During Admission	During Discharge

PLACE:

DATE:

Signature of Medical Officer.

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